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A QUANTITATIVE THEORY OF ORGANIC GROWTH (INQUIRIES ON GROWTH LAWS. II)*

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I. INTRODUCTION

WE MAY define organic "growth" as the measurable increase of an organic system in weight or length, distinguishing it thus from "differentiation" and "form development," which represent increase in organization. Excluding, as usual, increases through mere imbibition, depositing of reserve materials, etc., we may connect growth with the fundamental properties of living systems, and define it as follows: Growth is the measurable increase of an organic system, produced by its assimilation of materials obtained from its environment.

The phenomenon of growth is, of course, extremely heterogeneous and complex in its descriptive as well as in its causal analytic aspects. Even the chemical side of the process, the synthesis of building materials specific to the body, is obscure. Mitosis is as yet not to be explained in physico-chemical terms. But even if we possessed the knowledge of these matters, we should be very remote from an understanding of

* Pt. I: "Untersuchungen über die Gesetzlichkeit des Wachstums," *Roux' Archiv für Entwicklungsmechanik der Organismen*, 131, p. 613, 1934; here referred to as *"l. c."*

growth in higher organisms. For the growth capacity of an organism does not depend on the single cells, but on the organic system as a whole; obviously the latter determines whether, where, and when cells divide, increase, differentiate, how metaplasms and intercellular substances are formed, etc. Further, growth is widely dependent on external factors such as nutrition, temperature, life space, as well as such internal factors as hormones, progressive differentiation, change of water content, age, etc.*

Thus a rational mathematical treatment of growth processes seems at first rather hopeless, and critical investigators, conscious of the overwhelming complexity of the problem, are justly sceptical of such attempts. However, we may emphasize an important point. Various examples in physics and physiology (such as, for example, thermodynamics, the mechanics of continua, and material balance calculations in the physiology of metabolism) show that it is often possible to state statistical laws for complex phenomena as a whole, the single events of which are inaccessible to investigation. It may be that such procedures will be effective also with respect to organic growth. Growth, considered as a mere quantitative increase, is certainly a very abstract aspect and a statistical result of an innumerable multitude of single processes. Since on one side of an equation expressing some law of organic growth stands a very abstract term, i.e. the mere quantitative increase, not concerned with all the complexity of the intimate processes, with organization, form development, etc., therefore on the other side also may justly stand abstract statistical terms. Such a procedure may be compared with the laconism of a financial report, stating that the wealth of a country has increased by so many millions, since export was so many times greater than import. Behind these numbers lies, of course, an infinity of movements and of destinies. For granting these results, citizens and machines have worked in the most different manners, railway trains have transported goods inwards and outwards; a complex organization, from workshops to factories, trusts, and the whole state, has made the economic process possible, which has granted their living to millions of individuals, has destroyed other millions. But of all this the financial

* Further, a good approximation of the empiric curves by means of some formula does, as such, not mean very much; for very different analytical expressions may lead to similar curves and to a good approximation. Only if the mathematical analysis is based upon, and always related to, physiological experience, we can speak of a *rational* mathematical treatment.

report says nothing. In a similar way, the theory to be developed below gives a balance of transformations occurring in the living organism, but says nothing of the ways of these transformations, of their kind, number, detailed laws, organization, etc. The actual complexity is not overlooked or neglected, but the theory of growth, like the financial report, gives, for an abstract aspect of happenings (there the result of the economic processes converted into terms of money, here the mere quantitative increase of an organism), an abstract formulation.

2. FOUNDATIONS OF THE THEORY

For the foundation of a growth theory we may start with a consideration of stationary chemical states. Physical chemistry is in fact almost exclusively concerned with reactions in closed systems; but it is easily possible to indicate the conditions under which stationary chemical systems may be constructed, and the laws governing them. Obviously a chemical system $a \rightleftarrows b$ is stationary, if in unit time so much of a reacting substance a is continually introduced as is transformed into b, and if b is also continually removed from the system. If, however, the amount of reacting material a introduced per unit time is not equal to its originally transformed and disappearing amount, but, for example, larger, then obviously the amount of a must increase. Let E be the amount of introduced reacting material (a) per unit time, k the rate of transformation of a into b per unit time (according to the law of mass action), then the concentration of a increases (assuming that the reaction product b is continually removed) according to the following expression:

$$\frac{dm}{dt} = E - km \tag{1}$$

The concentration in the system is therefore at the time t:

$$m = \frac{E}{k} - \left(\frac{E}{k} - m_0\right) e^{-kt} \tag{2}$$

m_0 being the concentration at time $t = 0$. As can be seen from these equations, the concentration will increase asymptotically to a constant value characterized by the fact that the amount of material transformed is now equal to the influx (assumed constant). This maximal concentration (M) follows from (1) if we let t increase without limit: $M = \frac{E}{k}$. In such a way, the law of mass action can be extended to stationary systems, putting the problem to physical chemistry to construct and investigate systems of this type.

Now organisms may be considered, as is generally recognized, as stationary chemical states. Their growth results from the balance of the perpetual breaking down and building up of their component materials. The organism increases, if the latter is larger than the former; it becomes stationary if both processes are equal. Both processes are, of course, of immense complexity. The destruction of building materials manifests itself, chemically, in autolysis of surviving organs, which is acting certainly also *in vivo*; further, cytologically, in the destruction of cells and cellular systems (contents of glandular cells, destruction of erythrocytes, epithelium, epidermis, hairs, etc.). On the other side, the introduced alien materials serve partly, in energy metabolism, for supplying the energy requirements of the organism and partly they are transformed into building materials. There are close relations between metabolism of building materials and energy metabolism. It is, however, usual in physiology to consider nitrogen or protein metabolism as primarily concerned with metabolism of building materials while carbohydrates and fats supply the energetic requirements. Assimilation manifests itself as synthesis of building materials, cell enlargement, mitotic cell division, etc. For a quantitative analysis of growth it is, however, not necessary to consider the as yet inaccessible manifoldness of chemical and cytological events, but we may introduce constants which represent averages of all these processes of constructive and destructive metabolism.

The organism is, of course, a stationary system of a much more complex kind than that just considered. To obtain a starting point for a quantitative analysis, we must make an assumption as to the relation in which construction and destruction stand to the mass of the organism. As concerns the latter, the simplest assumption is that per unit time an approximately constant percentage of building materials is destroyed and lost, i.e. that destruction is proportional to the mass of the organism. This assumption corresponds to the usual physiological view. If K is a constant for destruction per unit mass, the destruction for the total mass is Km.

On the other hand, it may be assumed that construction of building materials is proportional to some surface. This assumption may be interpreted as signifying: (a) that synthesis is proportional to the resorption of nutritive material, and therefore to the magnitude of resorbing surfaces; (b) synthesis is dependent on the energy metabolism in which the necessary energies for synthesis must be gained; the latter, however, is in general proportional to a surface (Rubner's rule). We

may, therefore, tentatively define synthesis by a constant H (synthesized mass per unit surface); the total amount synthesized will be Hs. The actual increase (growth rate)* is then defined by the difference of these magnitudes:

$$\frac{dm}{dt} = HS - km \tag{3}$$

This expression was first used by Pütter (1920). Then the relative growth rate is:

$$\frac{dm}{mdt} = \frac{Hs}{m} - K \tag{3a}$$

This equation can be solved if we can make an assumption on the relation between m and s. Considering first the case of proportional or isometric (Huxley and Teissier, 1936) growth, in which the shape of the growing body remains geometrically similar, and surface and volume (or mass) can always be calculated by multiplication of the square, or cube, of any linear dimension with a constant ($s = l^2 p$, $m = l^3 q$), and therefore, relative growth rate of a linear dimension is $\frac{1}{3}$ of that of volume

$$\frac{dm}{mdt} = \frac{d(l^3 q)}{l^3 q\, dt} = \frac{3dl}{ldt},$$

we have:

$$\frac{dl}{dt} = \frac{Hsl}{3m} - \frac{Kl}{3}.$$

Since $\frac{sl}{m} = \frac{p}{q}$, we can write the last equation:

$$\frac{dl}{dt} = \frac{Hp}{3q} - \frac{Kl}{3},$$

and, setting $E = \frac{Hp}{3q}$, $k = \frac{K}{3}$, we have:

$$\frac{dl}{dt} = E - kl \tag{4}$$

* Growth rate is the increase (in length, mass, etc.) of the organic system in unit time $\frac{dx}{dt}$; relative growth rate is the increase in unit time relative to the existing value of the magnitude $\left(\frac{dx}{xdt}\right)$.

The solution of this equation is:

$$l = \frac{E}{k} - \left(\frac{E}{k} - l_0\right)e^{-kt}. \qquad (5)$$

The final length for t tending to infinity is $L = \frac{E}{k}$, wherefore (5) may be written:

$$l = L - (L - l_0)\, e^{-kt} \qquad (6)$$

For weight growth follows from (6) through a simple calculation:

$$m = [M^{\frac{1}{3}} - (M^{\frac{1}{3}} - m_0^{\frac{1}{3}})\, e^{-kt}]^3 \qquad (7)$$

Regarding the growth constants, as already said, $K = 3k$. Since the actual surface responsible for growth of an organism is in general unknown, we cannot determine directly the "true" value of H characteristic for the organism. For comparative purposes, however, synthesis may be referred to the fictitious surface $m^{\frac{2}{3}}$, as frequently used in physiology. Now since we have set $E = \frac{Hsl}{3m}$, in this case we have:

$$E = \frac{H\, m^{\frac{2}{3}}\, l}{3m} = \frac{H\, (l^3 q)^{\frac{2}{3}}\, l}{3 l^3 q} = \frac{H}{3q^{\frac{1}{3}}},$$

or $H = 3Eq^{\frac{1}{3}}$. This expression holds for the calculation of H from the E of the growth equation for any linear dimension; for the special case that we choose as linear dimension $l = \lambda = m^{\frac{1}{3}}$ [*i.e.* using equation (7)], $q = 1$, and $H = 3E$. The meaning of this is as follows: While k (destruction constant) has the same value for any linear dimension, E (except in the case of a spherical organism) is different in the divers spatial directions. If E be the same in the different dimensions, the organism, whatever its initial shape has been, would approach a spherical shape. Mathematically this relation is expressed through the fact, that E is the numerator, k the denominator of the expression $L = \frac{E}{k}$; transposing from one linear dimension to another, i.e. multiplying this expression with a constant indicating the proportion of the second to the first linear dimension, k remains unchanged while E becomes multiplied. We may say that, starting with any linear dimension of a non-spherical organism, the value of k calculated, following (4) or (5), is independent of the shape, and always equal to $\frac{K}{3}$. E, for any linear dimension,

however, depends on the shape of the organism (on the constant q with which l^3 must be multiplied for obtaining m). But the synthesis constant $H = 3Eq^{\frac{1}{3}}$ (or $3E$, if a linear dimension $m^{\frac{1}{3}}$ is chosen) is independent of the shape and the chosen, special dimension; it gives, therefore, an expression applicable for comparative purposes.

The methods of application of these formulas for calculating empirical growth curves are described in detail in the former paper (*l.c.*).

For the assumption that growth is conditioned by a counteracting of a furthering surface factor and an inhibiting mass factor, morphological as well as physiological arguments can be stated (for full discussion see *l.c.*). (I) Comparing the size of related animals (calcareous sponges, hydroids, Anthozoa, Turbellaria, dog races) it is found (Hesse 1927) that species possessing a rich development of intestinal (i.e. resorbing) surfaces grow much larger than such with poorer. (II) Comparing the growth curve of rod-like and spheroid bacteria (cf. Schmalhausen 1928), we see a fundamental difference. The former grow with almost constant relative growth rate $\left(\frac{dl}{ldt} = c\right)$, i.e. they show a simple exponential growth ($l = l_0\, e^{ct}$). On the contrary, in the growth of spheroid bacteria, relative growth rate decreases, and their linear growth is to be represented through equation (5). This striking contrast is to be explained as follows. Rod-like bacteria grow almost exclusively in length; since hereby not only the volume, but also very nearly the surface increases proportionally to the length, intake retains not only for unit surface, but also for unit volume, a constant value. Therefore, growth rate, as resulting from the difference between assimilation and dissimilation, is given by $\frac{dm}{dt} = Hs - Km \sim Hm - Km = cm$, yielding for length growth the formula $l = l_0\, e^{ct}$. In the case of spheroid bacteria, however, we have proportional growth in which the relation surface: volume is continually shifted in favor of volume; a progressive decrease of growth rate must result, and equation (5) must hold. In fact, the growth curves of rod-like as well as of spheroid bacteria follow with physical exactitude the equations derived from the assumption given above. Especially also the details of the growth curve prove that assimilation is proportional to the surface, dissimilation to the volume of the system. (III) It is known to physiologists that Rubner's rule does *not* hold for insects: while metabolism (O_2 consumption) is a surface

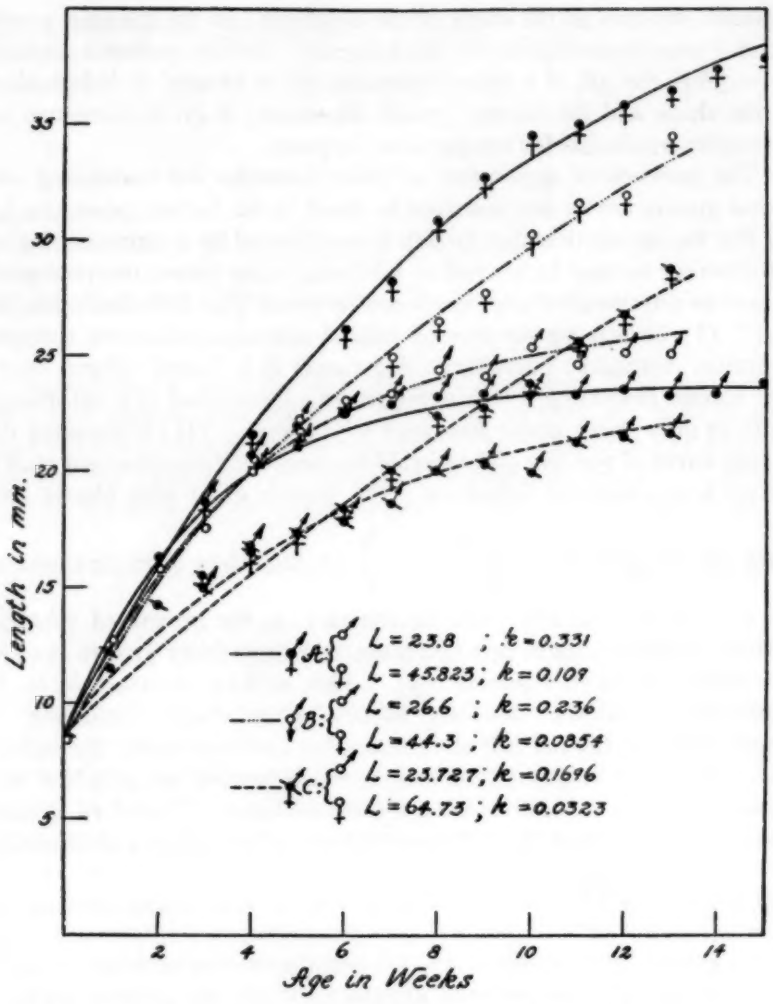

FIG. 1. LENGTH GROWTH OF THREE BREEDS OF LEBISTES RETICULATUS

Calculated curves with indication of observational values. First values: sexes not distinguished. Breed A and B: fed with living food; C: fed with Piscidin.

function in most animals (especially fishes and mammals), respiration increases in insects with the mass of the body. Corresponding to this metabolic peculiarity, the growth curve of insects shows an entirely different character from those of other animals: relative growth rates

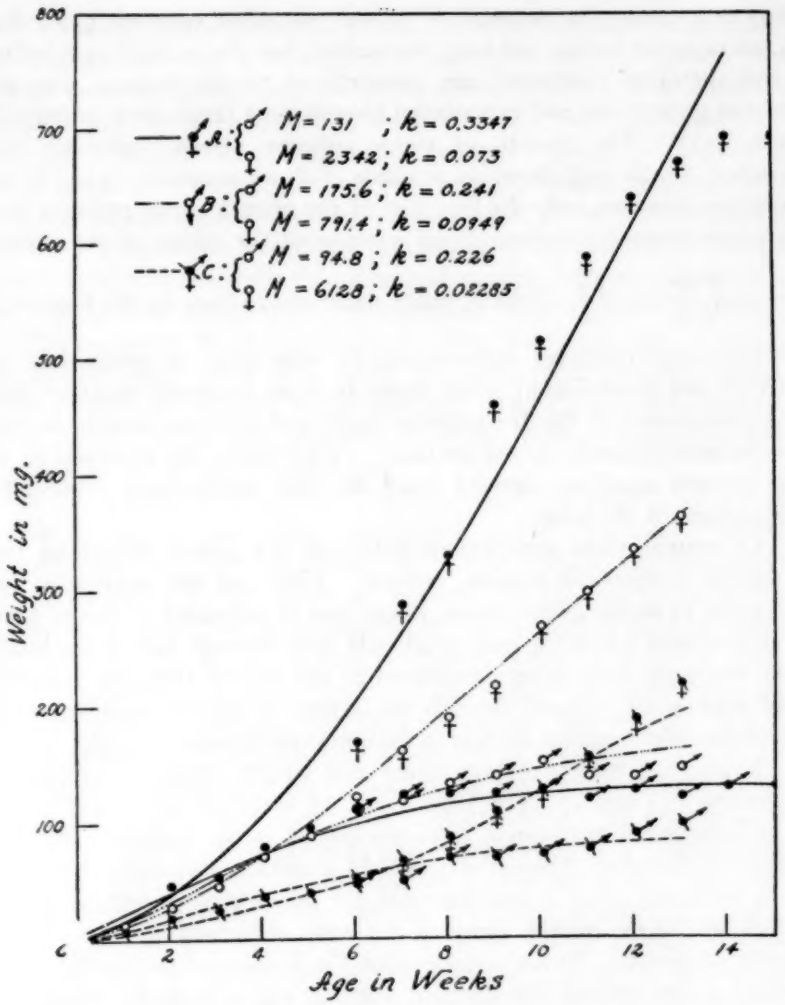

FIG. 2. WEIGHT GROWTH OF THREE BREEDS OF LEBISTES RETICULATUS

Indications as in Fig. 1.

remain constant, and growth is simply exponential. This fact also is a striking proof for our assumptions. In the other animals, energy metabolism (and therefore, according to our assumption, synthesis) corresponds to a surface; it must, therefore, in the growing organism fall short of destructive metabolism proportional to the volume, leading

thus to a continuous decrease of growth rate after equation (5). But if, as in insect larvae, not only destructive, but also energy metabolism (and therefore synthesis) are proportional to the volume, constant relative growth rate and exponential growth must result, as it happens in fact. (IV) The growth of tissue cultures (Buch Andersen, und Fischer, 1929) considered as a whole, follows equation (5). If we consider, however, only the first part of the growth curve, per unit time an approximately constant, linear increase of the radius of the culture occurs $\left(\frac{dr}{dt} = E\right)$. This is easily understood, since in the beginning surface—and therefore assimilation—is very large in comparison to volume and dissimilation, which latter becomes gradually stronger with the enlargement of the dissimilating mass; and it proves once more that assimilation depends on the surface. (V) Finally, the excellent fit of the growth equations derived from the said assumptions proves the correctness of the latter.

Of course, these assumptions hold only for animal organisms and somehow comparable organic systems. They are not applicable, for example, to higher green plants, where loss of substance is almost completely absent (occuring here practically only through fall of the leaf), and where the fact appears fundamental for growth that new assimilative organs are formed through the action of already existing ones. Here the considerations leading to the autocatalytic equation may apply.

Examining the curves for linear and weight growth according to equations (5) and (7) (cf. Figs. 1, 2) we find that the former shows a uniform levelling; growth rates are largest at the beginning and decrease continually. The curve of weight growth is essentially different; it is S-shaped, as it is characteristic for many growth curves, a fact which led to the widely accepted but very objectionable autocatalytic theory of growth. In our consideration the S shape of the weight curve results as an evident consequence without any subsidiary hypothesis. The point of inflexion of the curve (as is easily to be seen by formation of the second derivative) lies at $0{\cdot}2963M$ (while in the logistic curve it lies at $0{\cdot}5M$). Agreeing with these conclusions, the linear growth curves show, in general, a course corresponding to (5), the weight curves are S-shaped with the point of inflexion at about one-third of the mature weight. This is already a strong argument in favor of our point of view, and against the correctness of the logistic formula usually applied to growth phenomena.

Excluding certain cases (for example, rod bacteria and insect larvae, as mentioned above) it is a general feature of organic growth that growth rates continually decrease. It is spoken of often, in a somewhat mystical way as an unknown "growth tendency" of organisms. We may say, however, that growth and its gradual decrease is (at least in so far as the conceptions just developed hold) a simple consequence of the fact that the organism is a stationary system of the kind considered above. The theory of stationary systems gives an explanation for the growth curves and for the limitations of growth, both as expressed in the "rule of constant cell size" and in growth of multicellular organisms.

The numerical fit of the equations given above has been proved in the former paper (*l.c.*) by a series of examples of different organic systems, such as bacteria, tissue cultures, insects, other invertebrates, fishes, and amphibia. In fact, these formulae gave an excellent representation of the actual growth curves.

But it is possible to go even a step farther. In general we are, of course, not able to identify our constants H and K with specific physiological magnitudes, for the metabolism of a bacterium, a tissue culture, an animal cannot be considered as uniform. In the case of the animal organism, however, we may assume (of course, as first approximation and without prejudice to eventual further complications) that K represents the *Abnützungsquote* (Rubner), indicating the destruction and loss of building material (especially of protein nature) per unit of weight and time. The *Abnützungsquote* may be estimated by the determination of N excretion or protein loss in the starving animal. Comparing the values which have been calculated from the growth curve with those determined in physiological experiments, an excellent agreement was found (*l.c.*). This must be considered a very important verification of the theory. For through a purely mathematical analysis, based on the growth laws as developed above, we are able to calculate from the data of the growth of an organism (i.e. from a series of measurements of its weights or lengths), the magnitude of its destructive metabolism and these calculated values agree with the values directly measured in physiological experiments, i.e. in a way of investigation which has nothing to do with the observation of the growth process. Thus it is proved: (1) that these constants under discussion are not mathematically constructed entities, but physiological realities; and (2) the identity of constants found in two such different ways is a proof of the correctness of the theory. We can state that, at least in the best-investigated cases, the calculation of the growth curve contains

no arbitrary, but only empirically determinable constants. For, calculating with equation (6) or (7), we have only the two constants L (M) and k. L (M), the final value to which the growth curve asymptotically approaches, can, in principle, directly be read off from the growth curve; k is, as was shown, a constant determinable directly in physiological experiment. It will not be easy to find, outside of the realm of physics, a mathematical theory bearing this very important feature. Of course, in practice there is an amount of arbitrariness in finding those values for the constants which will give the best fit; but this arbitrariness is only one of the technique of curve-fitting, and does not touch the basic statement as made above.

3. GROWTH OF LEBISTES RETICULATUS (PETERS)

In this paper some new verifications and extensions of the theory will be given.

In our former publication a number of growth analyses of fishes were given. These examples, based on measurements of fishery biology, left the question open whether our equations are able to represent the *complete* growth process; for these measurements begin mostly with later periods of development, so that it may be questionable whether our equations hold for the important early postembryonic stages. To answer this question, the growth of the little viviparous cyprinodont, *Lebistes reticulatus,* was investigated. This animal was chosen firstly because it seemed reasonable to study a viviparous fish, in order to eliminate the embryonic stages, which are not to be considered onesidedly from the point of view of quantitative increase. The newborn have, however roughly, the shape of the adults, so that growth may be interpreted approximately according to our formulas. Secondly, the growth of *Lebistes* is interesting in regard to the very great sexual differences in size, the females reaching about double the length and a multiple of the weight of the males. Thirdly, data being given, as yet, only for slow-growing fresh-water fishes, the growth of a fast-growing warm-water fish is interesting.

The fishes were kept at a practically constant temperature of 24° C.; they were fed partially with living food (copepods in the earliest stages, later *Tubifex* with occasional addition of daphnids), partially with Piscidin. Every week, length and weight were measured. The sexes can be distinguished at an age of about three weeks.

The observed values and calculated growth curves (linear and weight growth) for three different breeds are represented in Figs. 1 and 2. The theoretical curves are calculated by method II 1a and II 2a, *l.c.* page 627. As can be seen, different breeds show considerable differences in their growth curves (and, of course, of growth constants), even on equal diets. This is easily to be understood, since *Lebistes reticulatus* of our aquaria is certainly a very impure race, so that there are certainly genetic differences. (Breed 1 was raised from a stock kept a long time in pure breeding in II Zoologisches Institut der Universität Wien while the females from which breeds 2 and 3 were obtained were

TABLE 1

Length growth of Lebistes reticulatus (Breed A)

AGE IN WEEKS	V ♂	V ♀	$M \pm m$ ♂	$M \pm m$ ♀	v ♂	v ♀
0	8.0- 9.25		8.75±0.184		4.71	
1	10.0-12.5		11.5 ±0.195		6.36	
2	15.0-18.0		16.3 ±0.258		5.94	
3	17.5-20.0		18.6 ±0.187		3.64	
4	19.50-22.5	20.50-29.0	20.6±0.218	21.6±0.422	3.81	5.92
5	20.00-22.0	21.00-24.5	21.1±0.154	22.0±0.339	2.43	4.92
6	21.00-24.5	24.50-29.5	22.5±0.237	26.1±0.449	4.60	5.96
7	21.50-24.5	26.50-31.5	22.9±0.267	28.7±0.458	4.4	5.85
8	22.25-24.0	28.75-33.5	23.2±0.144	30.8±0.369	2.77	4.15
9	22.00-25.0	30.00-35.5	23.4±0.230	32.7±0.397	3.93	4.55
10	22.00-25.0	32.00-37.0	23.2±0.241	39.5±0.461	3.89	4.88
11	22.00-24.5	30.00-37.5	23.3±0.204	35.0±0.607	3.23	6.02
12	22.50-25.0	34.00-38.0	23.6±0.183	35.7±0.299	3.31	3.22
13	21.00-24.0	34.00-39.5	23.3±0.243	36.4±0.481	4.17	4.94
14	21.50-25.0	35.00-41.5	23.4±0.251	37.4±0.561	4.03	4.97
15	22.00-25.0	35.00-42.0	23.8±0.210	37.9±0.543	3.19	5.35
16	22.00-27.0	35.00-42.0	24.2±0.268	38.0±0.504	4.84	5.14

V = total spread
M = arithmetic mean
m = standard error of $M = \frac{\sigma}{n^{\frac{1}{2}}}$
v = variation coefficient $= \frac{100\,\sigma}{M}$

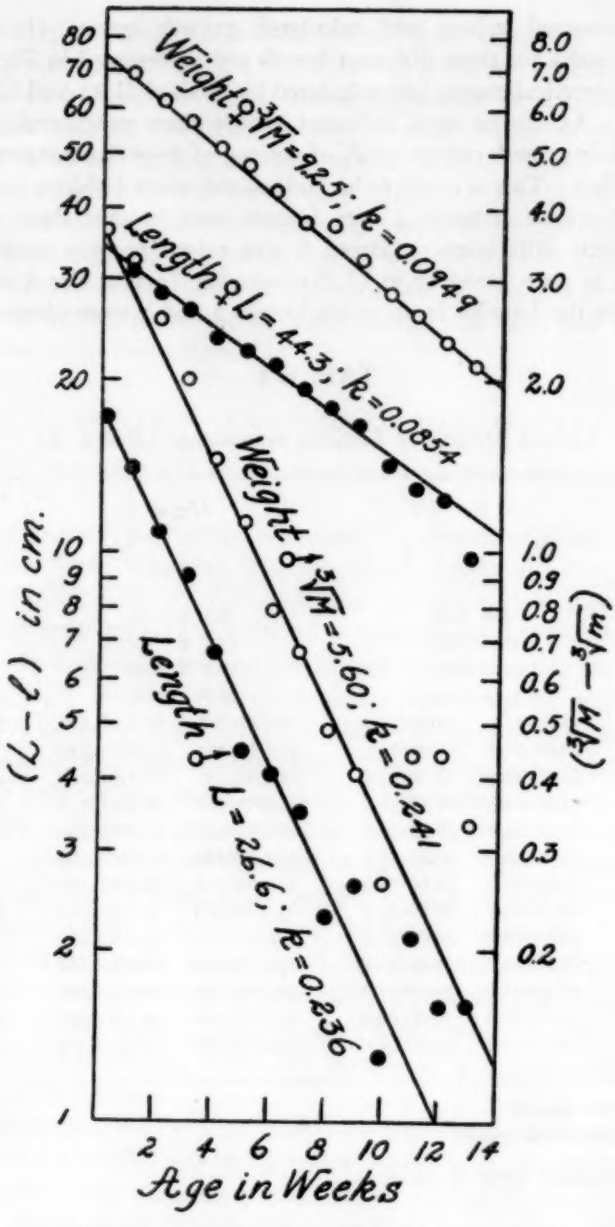

FIG. 3. LENGTH AND WEIGHT GROWTH OF LEBISTES RETICULATUS (BREED B)

Semilogarithmic plot of $(L-l)$, or $(M^{\frac{1}{3}}-m^{\frac{1}{3}})$ against t.

the usual commercial articles). Significant are the differences between breeds fed with living and artificial food; the poorer nutrition of the latter seems to lead to a weaker metabolism, showing itself in lower growth constants. From the statistical treatment of the material (Table 1) not very much is to be learnt. The variability (variation coefficients) seems to be a little higher in females than in males; but the difference is not significant. The material is, however, very small for statistical purposes (about 20 in each sex) since it seemed, in regard to the differences between different breeds, not permissible to treat the individuals of the different breeds together.

It is possible to fit the whole growth curve of *Lebistes* by our formulas (5) and (7). The fit may be illustrated by Fig. 3 and Table 2 (after method II 2a). From formula (5) it is seen that

$$\log (L - l) = \log (L - l_0) - kt \log e,$$

i.e. the differences between final length and actual length (or cube roots of weights), plotted semi-logarithmically against time, must give a straight line, as they do actually to a good approximation.

TABLE 2

Growth of Lebistes reticulatus (Breed B)

AGE IN WEEKS	LENGTH IN MM. OBSERVED		LENGTH IN MM. GRAPHICALLY CALCULATED		WEIGHT IN MG. OBSERVED		WEIGHT IN MG. GRAPHICALLY CALCULATED	
	♂	♀	♂	♀	♂	♀	♂	♀
0	9.4		9.4*		7.6		7.6*	
1	12.6		12.6*		13.0		16.8*	
2	15.7		15.5*		28.0		33.6*	
3	17.5		17.7*		46.7		51.8*	
4	19.9	20.5	19.8	19.8	71.4		72.8*	
5	22.1	21.8	21.2	21.8	89.3		94.8*	
6	22.5	23.0	22.4	23.5	111.2	121.7	105.2	130.3
7	23.2	24.9	23.3	25.3	120.0	162.5	117.6	161.9
8	24.3	26.4	24.0	26.8	133.3	190.0	129.5	197.1
9	24.0	27.7	24.5	28.3	140.0	217.5	138.2	232.6
10	25.3	30.2	25.0	29.5	152.5	266.7	146.4	268.3
11	24.5	31.5	25.3	30.7	137.5	296.7	152.3	298.5
12	25.0	31.9	25.6	31.8	137.5	333.3	156.6	331.4
13	25.0	34.5	25.8	32.8	146.0	363.3	161.0	362.09

*Means of calculations for males and females.

4. MAMMALIAN GROWTH

The analysis of mammalian growth encounters special difficulties. Especially the careful work of Saller (1927) shows how complex the situation is, and that most of the available observation series do not comply with the demands indispensable for a deeper analysis.

The first demand for a successful analysis of mammalian growth is a *suitably large material.* For individual variability is here, even in uniform breeds, extraordinarily high; so, for example, according to Saller variation in newborn mice is between 0.75 and 1.95 g., in 5-weeks-old mice between 5.5 and 16.1 g., so that the lightest individuals have only a third of the weight of the heaviest one. We are therefore not entitled, given only a small number of individuals, to consider the arithmetic mean as characteristic.

Further differences of outer and inner growth factors are of considerable importance. As outer factors, of course, different *nutrition, temperature,* possibly *seasonal cycles* are to be considered. As regards inner growth factors, the great and unanalyzable *complexity* of the fundamental processes; the influence of *genetic factors* (as well on the growth curve as a whole as on the final weight reached); and eventually also the influence of a *senescence factor* are to be mentioned. The most important difficulty for the analysis of mammalian growth is, however, the existence of *growth cycles.* It is to be emphasized that the attempt of analysis is rather hopeless, without an exact knowledge of the temporal distribution of those cycles, which is only granted where we have available especially carefully collected observational material.

Mammalian growth cannot be considered as uniform. For the outer conditions of growth as well as the inner system conditions change in the course of development: birth, ablactation, sexual maturity are connected with important changes of total metabolism which must affect the growth curve. These changes are obviously intimately connected with changes in the hormonal system. In the case of man e.g., after Biedl, firstly adrenals and related parts of sexual glands dominate; then an increase of thyroid activity occurs, which is connected with decrease of anabolism; in the period of first stretching the anterior lobe of the hypophysis acts as growth regulator; puberty is characterized by hyperactivity of thyroid and hypophysis, connected with functional atrophy of the thymus. Finally, new metabolism-regulating glands, such as the adrenals, the insular organ, and the middle lobe of the hypophysis influence metabolism. Corresponding to the changes of conditions, the

TABLE 3

TABLE 3

Absolute and relative growth rates in weight growth of male white mice

AGE IN WEEKS	STIEVE 1923		ROBERTSON 1916		ROBERTSON 1926		SALLER 1930		KOPEČ 1932	
	$m_o - m$†	$\frac{100(m_o - m)}{m}$	$m_o - m$	$\frac{100(m_o - m)}{m}$	$m_o - m$	$\frac{100(m_o - m)}{m}$	$m_o - m$	$\frac{100(m_o - m)}{m}$	$m_o - m$	$\frac{100(m_o - m)}{m}$
1	1.25	71.4	2.08	169.1	1.52	103.4	1.72	137.6	2.78	222.4
2	1.33	44.3	1.83	55.3	2.10	70.2	1.67	56.2	2.51	62.3
3	0.77	17.8	(3.62)*	**60.4**	**2.90**	**57.0**	1.23	26.5	2.22	33.3
4	**1.70**	**33.3**	(3.62)	41.3	1.52	19.0	**2.16**	**36.8**	**3.37**	**88.5**
5	1.03	15.1	0.07	00.6	1.76	18.5	**2.75**	34.2	2.81	23.2
6	1.09	13.9	3.13	25.1	1.70	15.1	2.07	19.2	2.76	18.5
7	1.28	14.3	2.50	16.0	1.94	15.0	(1.04)‡	8.1	2.46	14.0
8	0.90	8.8	1.28	7.1	1.74	11.7	....	.....	1.53	7.6
9	1.40	12.6	1.27	6.6	1.40	8.3	....	.....	0.83	3.8
10	0.80	6.0	0.56	2.7	0.63	3.5	....	.....	0.67	3.0

* Figures in parentheses: computed values, cf. text.

‡ 49th day.

† m, m_o: weights at subsequent times.

growth curve shows segments, with discontinuities of its slope, and mathematical analysis must account for this fact in terms of *subdivision* of the growth curve.

We shall confine ourselves to postembryonic growth and choose as first example that of the white mouse. According to Saller (1927), two growth cycles are here to be considered as well established. During the first three weeks, growth rates decrease; then follows a rise, culminating about the fourth week, and finally a new, continuous decrease lasting to the end of the progressive phase. Table 3 gives absolute and relative growth rates, calculated by the method of Minot*; the second peak of absolute and relative growth rates is shown in heavy figures. About the time of the break between the first and second cycles (at an age of about 19 days) eye-opening, ablactation, and consumption of alien nutrition occurs. Likewise about this time processes are beginning which lead in the subsequent period (3rd-5th week) to the final development of sexual organs: the ripening of sexual glands begins (Saller, 1932, p. 620) about the 16th day, ripe spermatozoa appearing on the 35th-42nd day; obviously also the development of sexual differences in weight (from the 16th day, Saller) is connected with the ripening of sexual glands. About the 3rd week, therefore, a fundamental displacement of the total metabolism, arising from hormonal changes, occurs. The assumption of a second break (about the 5th week) and therefore of three cycles (1st to 3rd extrauterine weeks; 3rd to 5th week; 5th week to the close of the progressive phase), as made by Saller, seems to be superfluous in regard of the data given above; Saller himself is not quite clear on the position of this second break; after having at first set it on the 35th-42nd day, in his later publication (1932), he assumes that it may fall on a later date (about the 49th day), without being able to fix it exactly.

With respect to these cycles, Table 4 gives the observational and calculated data for the growth of the white mouse (after the careful measurement of Stieve, 1923). As a comment on this calculation, the following points may be mentioned. A growth curve, which is not

* The calculation of relative growth rates by Minot's formula

$$\frac{dx_t}{x_t dt} = \frac{(x_{t_1} - x_t)}{(t_1 - t)x_t}$$

does not give the exact values (cf. *l.c.*); in this connection, however, its application is legitimate since it is used only for purposes of comparison.

TABLE 4

Growth of white mouse (♂) after Stieve 1923

AGE IN WEEKS	WEIGHT IN GRAMS		AGE IN WEEKS	WEIGHT IN GRAMS	
	Observed	*Calculated*		*Observed*	*Calculated*
0	1.75	1.75	16	17.70	17.8
1	3.00	3.02	17	18.20	18.4
2	4.33	4.30	18	18.70	18.9
3	5.10	4.95	19	19.20	19.4
4	6.80	6.17	20	19.60	19.8
5	7.83	7.41	21	20.00	20.2
6	8.92	8.64	22	20.50	20.5
7	10.20	9.84	23	21.00	20.8
8	11.10	11.0	24	21.20	21.0
9	12.50	12.1	25	21.50	21.3
10	13.30	13.1	26	21.70	21.5
11	14.30	14.1	27	22.00	21.7
12	14.90	14.9	28	21.50	21.9
13	15.60	15.7	29	21.80	22.0
14	16.30	16.5	30	21.60	22.2
15	17.00	17.2	..	....	...

I. $m = [2.132 - (2.132 - 1.205)e^{-0.3020t}]^3$
II. $m = [2.855 - (2.855 - 1.205)e^{-0.121t}]^3$

subdivided into cycles, and which runs according to our equation (4), shows an increase of the absolute growth rates (till about one-third of final weight), followed by a decrease; the relative growth rates (calculated e.g. by Minot's method) are continuously decreasing. Assuming two growth cycles, in accord with the observations referred to above, the first lasting from birth to the second week, the second thenceforward to the stopping of growth, we are able to reproduce (1) the total growth curve with considerable closeness, and (2) also the details of the process, the peculiar changes in absolute and relative growth rates, with a very good approximation (Fig. 4). We think that there is, as yet, no example where analysis of a complex growth process with all details is achieved with a comparable elegance. Comparing for example the monstrous superposed curves Robertson used to reproduce the growth curve of the white mouse (1926) with our procedure, which

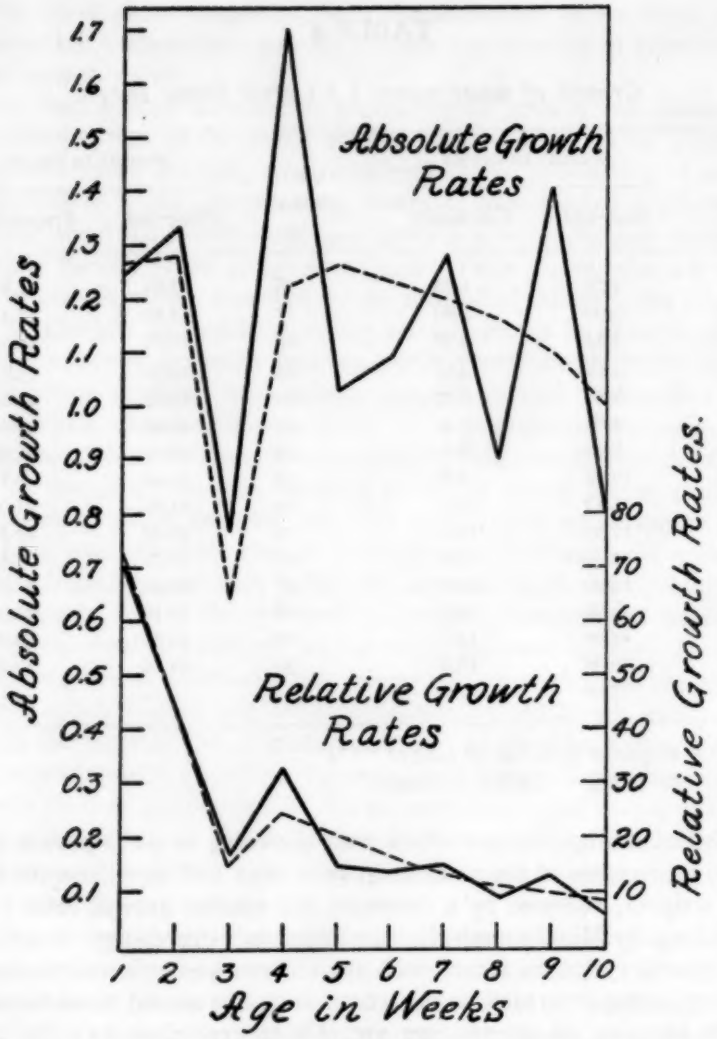

FIG. 4. ABSOLUTE AND RELATIVE GROWTH RATES IN WHITE MOUSE, OBSERVED AND CALCULATED

Observed:————; Calculated: - - - - - - - - -.

comprehends by means of two simple and formally identical equations the complex process with all fineness, the progress made in the present analysis may not be denied. Although bearing in mind that, of course,

a complicated curve like that of growth may well be approximated by quite different functions, we think that, from the numerous attempts to give a mathematical expression for the growth curve by means of very different functions, none has succeeded in a manner so simple, satisfying, and in agreement with physiological experience; and that, in view of the different efforts already made, it is not probable that a better representation will be found in future.

In treating, as a second example, the growth of the white mouse according to Robertson, we shall, however, make no effort to give an analysis covering all details (the possibility of which is already demonstrated by the foregoing), for the simple reason that the observational material, because of deficiencies in the experimental technique, does not justify it. Nevertheless, Robertson's data have a certain historic importance, and we want to show, therefore, that they may be expressed better by means of our simple equation, than by his own complicated system of equations.

The technical deficiencies mentioned are as follows:

"The actual observations of Robertson begin only at an age of 5 weeks; the data for younger animals Robertson has taken, without considering sexual differences, from the publication of Robertson and Cutler (1919). Now Robertson and Cutler give weights of their animals (whose total number, without regard to sexual differences, is only 18 in the lowest counting) only to the 20th day of extrauterine life; the value for the 3-weeks-old animals is computed by Robertson (1926, p. 478) under the supposition that the weight increase from the 20th to the 21st day will be exactly the same as from the 19th to the 20th day; the value for the 4-weeks-old animals was interpolated as mean between the values for 3 and 5 weeks. Obviously through this estimation a considerable arbitrariness is introduced into Robertson's data" (Saller, 1927, p. 478 ff.).

It appears, therefore, that Robertson's data do not allow an exact analysis of the growth cycles, concerning just the first 3 weeks, because the values for the 3rd and 4th weeks are dubious, and two different observational sets must not be joined. The part of growth which is free from those experimental deficiencies, and therefore worth being analyzed, is that from the 5th week on. As we see, the simple application of the method of least squares following from our equations (*l.c.*) gives an excellent approximation, so that our single formula represents better the part of the growth curve free from experimental faults, than Robertson's four superposed curves (Table 5).

In regard to our calculation of Robertson's data, it is, however, to be mentioned that it is cut in the 30th week, and that it assumes a

TABLE 5

Growth of the white mouse (♂) after Robertson 1926

AGE IN WEEKS	OBSERVED WEIGHT IN GRAMS	PROBABLE ERROR	CALCULATED WEIGHT IN GRAMS	DIFFERENCE BETWEEN CALCULATED & OBSERVED VALUES	
				After Bertalanffy	*After Robertson*
5	11.27	±0.29	11.27	0.00	−0.02
6	12.97	±0.32	13.09	+0.12	+0.12
7	14.91	±0.32	14.78	−0.13	+0.06
8	16.77	±0.30	16.32	−0.45	−0.21
9	18.17	±0.28	17.70	−0.47	−0.41
10	18.80	±0.28	18.93	+0.13	−0.09
11	19.80	±0.30	20.01	+0.21	−0.27
12	21.16	±0.30	20.96	−0.20	−0.87
13	21.80	±0.29	21.79	−0.01	−0.79
14	22.46	±0.29	22.51	+0.05	−0.77
15	22.84	±0.30	23.13	+0.29	−0.52
16	23.19	±0.31	23.66	+0.47	−0.28
17	23.83	±0.31	24.11	+0.28	−0.38
18	24.51	±0.31	24.50	−0.01	−0.57
19	24.66	±0.30	24.84	+0.10	−0.27
20	24.76	±0.30	25.12	+0.36	+0.03
21	24.97	±0.28	25.37	+0.40	+0.18
22	25.17	±0.26	25.58	+0.41	+0.29
23	25.65	±0.27	25.75	+0.10	+0.10
24	25.69	±0.32	25.90	+0.21	+0.31
25	26.06	±0.34	26.03	−0.03	+0.16
26	26.48	±0.35	26.14	−0.34	−0.07
27	26.48	±0.32	26.23	−0.25	+0.11
28	26.68	±0.29	26.31	−0.37	+0.07
29	26.33	±0.29	26.38	+0.05	+0.56
30	26.77	±0.31	26.43	−0.34	+0.25

Normal equations:* $24E - 66.9324k = 0.8056$

$66.9324E - 187.6534k = 2.08289$

$m = [2.9904 - (2.9904 - 2.2420)e^{-0.1088t}]^3$

(Time of calculation begins with 5th week as first considered date).

* The coefficients E and k in equation (4) can be determined, by a least squares method, through the normal equations (n = number of investigated cases):

(1) $nE - \Sigma lk = \Sigma \frac{dl}{dt}$

(2) $\Sigma lE - \Sigma l^2 k = \Sigma l \frac{dl}{dt}$

For weight, instead of l, $m\dot{l}$ is to be taken.

smaller final weight than found actually in the latest observed times. According to Robertson, there is a "linear increment." Our procedure is based on the fact that this linear increment is very dubious. The weights of Robertson's individuals are almost stationary during the 26th-30th weeks; in the 29th week there is even a decrease in weight, a fact which suggests that growth is really finished. (Even more clearly the maximal weight is reached by Stieve's individuals during the 26th week). In a later time, "linear increment" appears, lasting till a few weeks before natural death. "This weight increment, lasting till an age which corresponds almost to an age of 60 years of man, cannot be considered as a real growth increment." (Hoesslin, 1930, p. 608). This linear weight increment of Robertson's animals is to be considered as "fattening after the close of the progressive phase" (Sallet, 1927, p. 534), caused by an especially rich food in the breeding, and not as real growth which alone is accounted for in our considerations.

As a second example, we analyze the growth of the rat. We must assume here also the existence of growth cycles, which is already men-

TABLE 6

Growth constants of the white rat (♂). *Data of King 1915 (from Donaldson, 1923, Table 120)*

AGE IN DAYS	WEIGHT IN GRAMS	k	
13	17.2	.....	
30	48.5	.01635	calc. with: $M^{\frac{1}{3}} = 7.0$
60	122.9	.01655	
90	184.8	.01585	
120	232.2	.....	
151	244.8	.00832	calc. with: $M^{\frac{1}{3}} = 6.9$
182	258.4	.00731	
212	268.0	.00665	
243	279.7	.00683	
273	280.9	.00590	
304	296.1	.00690	
334	300.8	.00668	
365	306.1	.00672	
395	314.1	.00760	
425	312.2	.00617	
455	323.9	.....	
485	326.0	.....	

tioned by different observers, and which is clearly seen also in our method of calculation, the values of k, as calculated with a chosen final value (method II 2b, *l.c.*) being remarkably different before and after the 61st day (Table 6). This time corresponds nearly to the beginning of sexual maturity. The course of length growth corresponds to that

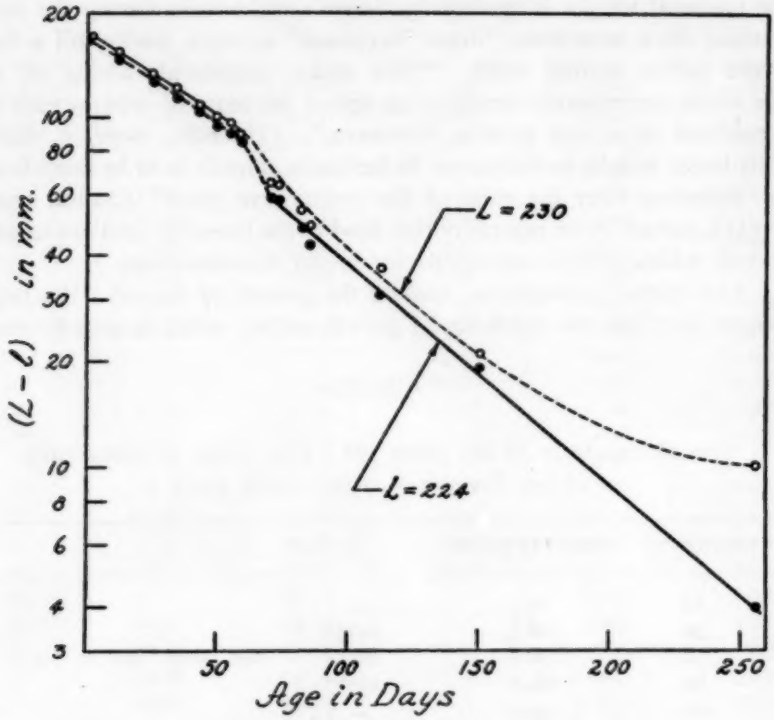

FIG. 5. GRAPHICAL ANALYSIS OF LENGTH GROWTH OF MUS DECUMANUS (WITHOUT TAIL), AFTER DONALDSON 1909 (FROM PRZIBRAM 1913, TAB. IX)

Semilogarithmic plot of $(L - l)$ against t. Showing the two growth cycles, with $L = 230$ in the first, $L = 224$ in the second cycle.

of weight growth. In graphical analysis (Fig. 5), the subdivision of the growth curve is also obvious. While, assuming a final length $L = 224$, the values following the 70th day form a very good straight line, with this final length the values from birth to the 58th day are arranged in a convex curve. This means that the assumed final value is too small.

TABLE 7

Length growth of Mus decumanus (without tail), after Donaldson 1909. (From Przibram, 1913, Tab. IX)

AGE IN DAYS	LENGTH IN MM.	$L-l$ OBSERVED (Final length $L=230$)	$L-l$ OBSERVED (Final length $L=224$)	$L-l$ GRAPHICALLY CALCULATED	LENGTH IN MM. GRAPHICALLY CALCULATED
0	59	171	..	171	59
12	76	154	..	151	79
25	98	132	..	132	98
34	109	121	..	120	110
43	121	109	..	109	121
49	130	100	..	103	127
55	134	96	..	96	136
58	141	89	..	93.5	136.5
70	165	...	59	59	165
73	166	...	58	58	166
82	176	...	48	49.5	174.5
85	181	...	43	48	176
112	193	...	31	31	193
150	209	...	19	18	206
256	220	...	44	4	220

TABLE 8

Comparison of growth constants in the rat

	L		E		k		H		K	
CYCLE	1	2	1	2	1	2	1	2	1	2
Length	230	224	2.415	3.308	0.0105	0.01477	0.217	0.298	0.0315	0.0443
Weight	6.84	6.558	0.075	0.0977	0.011	0.0149	0.226	0.293	0.033	0.0447

L, E, k directly obtained from the growth equations, $H = 3Eq^{\frac{1}{3}}$, being for length in this $q = 2.7 \times 10^{-5}$. As is to be seen, the growth constants H and K come out equal in magnitude from the independent calculations of linear and weight growth.

Choosing now for the first period the correct final value $L = 230$, all values completely fall into a straight line. The concordance between observed and (graphically) calculated values is very good here also (Table 7). Just so is the concordance between the values of H and K, derived independently from weight (arithmetical method) and length measurements (graphical method) (Table 8). A calculation of the well-known data of Donaldson is given in Table 9.

TABLE 9

Growth of the white rat (♂) after Donaldson

AGE IN DAYS	WEIGHT IN GRAMS		
	Observed	*Calculated*	
0	5.4	5.4	$m = [320^{\frac{1}{3}} - (320^{\frac{1}{3}} - 5.4^{\frac{1}{3}})e^{-0.0114t}]^3$
10	12.2	12.0	
21	21.2	22.0	
31	31.8	33.5	
40	42.2	45.3	
55	68.5	67.1	
61	81.7	76.2	
70	100.8	102.7*	
82	135.0	136.0	$m = [282^{\frac{1}{3}} - (282^{\frac{1}{3}} - 5.4^{\frac{1}{3}})e^{-0.0081t}]^3$
92	152.3	152.2	
109	168.8	167.0	
112	183.8	180.7	
124	197.3	195.2	
138	209.7	209.9	
150	225.1	220.8	
164	231.4	231.5	
178	239.4	240.5	
216	252.9	257.9	
256	265.4	268.7	
365	279.0	279.4	

* Mean value of the calculations for the 1st and 2nd cycles.

5. A CALCULATION OF MAN'S PHYSIOLOGICAL PROTEIN MINIMUM FROM HIS GROWTH CURVE

It was mentioned above that the theory succeeded, in a number of cases, in giving a theoretical deduction of the intensity of catabolic

protein metabolism, yielding the same values as were found in physiological experiments for protein excretion, which, as a first approximation and in accord with usual physiological views, is considered as a measure of the former.

However, the usual techniques do not give a complete survey of actual protein metabolism, especially in mammals.

(I) The most important fractions of the *Abnützungsquote* (Rubner), resulting from the "wearing off" of the building materials in living organisms, are: (a) wearing off of epidermis, nails, hair, feathers, etc.; (b) perishing of epithelia of inner organs, N loss through sperma, ova, menstrual blood, etc.; (c) loss of body substance through gland secretion, especially glands of the intestinal tract; (d) perishing of blood. In usual practice only N excretion in faeces and urine is determined; therefore, N loss in epidermis, hairs, feathers, sweat, mucus of respiratory organs, sperma, menstrual blood, etc., is neglected. The accumulation of creatin, creatinine, and ammonia in the muscle substance of starving animals shows that their actual catabolic protein metabolism is greater than N excretion.

(II) As is emphasized especially by Kestner, a re-use of already decayed body materials occurs. If the organism gets less nitrogen than it needs, metabolism and even to a certain measure growth continues. Obviously proteins arising from cell decay circulate in the blood, in the form of amino-acids, and are captured by the cells and resynthesized to proteins. Resynthesis of amino-acids liberated by cell decay is an imposing phenomenon, e.g. in the mighty development of sexual organs of salmon starving during up-stream migration, at the expense of vanishing muscles; but it is probably a general phenomenon. If, for example, after Ellenberger and Hofmeister (Bertram and Bornstein, 1928), the protein requirements of intestinal glands in N-free diet represent already an amount corresponding to the total N requirement of man, obviously the requirements must be satisfied by a repeated use of proteins. Therefore, N excretion is no exact measure of actual protein metabolism, since only a part of catabolized cell material is actually disintegrated to ammonia and urea and leaves the body, while another part is only reduced to amino-acids and resynthesized.

(III) Obviously this lack in experimental determination must be significant especially in experiments of short duration. In long experiments (e.g. several weeks of starvation in man) we may reckon, in some measure, that, firstly, the destructed protein material leaves the body through faeces and urine, and that, secondly, though also here a

restablishment of already catabolized protein occurs, the new-formed protein is itself disintegrated and so on, so that, in spite of these uncontrollable events in intermediate metabolism, finally an amount of nitrogen corresponding to the actual catabolism leaves the body. But this is not granted in short experiments (in starving a mouse for some days, after which it dies). Possibly here also the size of the animal plays a rôle, since, in the rebuilding of amino-acids of the blood, some re-resorption process must be involved whose intensity will depend on the magnitude of resorbing surfaces, the latter being relatively larger in small animals.

(IV) In protein metabolism some different concepts or physiological magnitudes are to be distinguished: (a) *N excretion in complete starvation* (in adult man 7-12 g. N daily, corresponding to 45-75 g. protein). N excretion in complete starvation is, however, as is well known, not the smallest possible. (b) *Minimal N excretion* occurs if an organism, in complete N starvation, is nourished abundantly with carbohydrates; then a far lesser protein excretion is found. (c) *Minimal N equilibrium*, the amount of excreted protein being equal to that of incoming, comes out, therefore, in an ample carbohydrate diet, and lies considerably below protein excretion in complete starvation. Thus man may already be in equilibrium with 0.4-0.6 g. protein per kg., i.e. with 25-45 g. total daily (Hindhede and other diet reformers). (d) Finally *physiological protein minimum* is the amount of protein which, all energetical needs of the organism being satisfied by non-protein materials, is necessary for the conservation of the organism at complete efficiency. It is usually assumed, for man, equal to 1 g. protein per kg. body weight daily, corresponding nearly to protein excretion in complete inanition. Therefore the question arises whether N excretion in complete inanition, or the much smaller minimal N excretion on carbohydrate diet, represents the better measure of *Abnützungsquote*. Opinions are divided. It seems to us that the first assumption is to be preferred. Considering the best-known example—man—it is rather commonly agreed that he cannot dispense, in the long run, with an amount lying considerably below the physiological protein minimum. "Finishing such experiment and allowing the man more protein, the very strong nitrogen retention in the body shows how great the nitrogen need actually was" (Kestner). The experiments mentioned belong therefore, after Kestner, to a stage where the amino-acids arising from destruction of cell components are captured by the cells and resynthesized to proteins. On the other hand, if minimal N excretion would represent the *Abnützungsquote* between the latter and the generally accepted physiological protein minimum there

would be a striking discordance. Thus, for example, Bertram and Bornstein consider minimal N excretion, or minimal N equilibrium, a measure of tissue catabolism. But, on the other hand, they write: "After a decade's investigations, the magnitude of practical protein minimum, all energetic needs being supplied by non-protein nourishment, seems to be 80 g. for an organism weighing 70 kg., i.e. it amounts to one-half times the protein amount required for supplying the minimal N equilibrium." It is hardly intelligible why, if for supplying the tissue catabolism only about 20 g. are necessary, and if the energy needs of the organism are supplied completely by non-protein nourishment, a protein amount three and one-half times larger is necessary, or even desirable.

On the other hand, the "protein-sparing" action of carbohydrates, i.e. the phenomenon that the organism being overfed with carbohydrates, gets along with a smaller protein supply than what could correspond to the excretion in starvation, is probably due to the fact that, to a certain extent at least, protein synthesis from N-free acids and ammonia is possible in the animal organism. We may mention, e.g. some classical experiments: feeding a dog with γ-phenyl-α-keto-butyric acid, $C_6H_5 \cdot CH_2 \cdot CH_2 \cdot CO \cdot COOH$, according to Knoop the corresponding amino-acid $C_6H_5 \cdot CH_2 \cdot CH_2 \cdot CH(NH_2)COOH$ is excreted; according to Embden, from pyruvic acid, or oxyphenyl-pyruvic acid, when conducted through dog's liver, alanin, or tyrosin is formed. Since carbohydrates yield pyruvic acid, and, on the other hand, ammonia, formed through protein-decay, is available in the organism, it is intelligible that, when through abundant carbohydrate import larger amounts of pyruvic acid are available, from these components new protein may be synthesized. A strong argument in favor of the assumption that the protein-saving action of carbohydrates is based on the possibility of protein-resynthesis is given by the fact that, feeding fats in an amount corresponding to the calory requirement, protein destruction is scarcely less than in complete inanition; for fats are β-oxydized, their derivatives do not suit, therefore, for synthesis of the α-animo-acids as existing in the organism, while a corresponding amount of carbohydrates lowers considerably the N excretion. The last-mentioned fact, reduction of N excretion on carbohydrate diet, is also easily understood from these considerations, since nitrogen, arising from catabolized cells, etc., is not excreted but retained, for forming anew amino-acids and proteins.

We may conclude, therefore, that the best available measure of tissue catabolism is protein loss in inanition, these determinations being trust-

worthy especially in the case of long experiments where the errors mentioned above (namely, the retention of actually catabolized proteins, their resynthesis, and protein synthesis from derivatives of carbohydrates and ammonia) are less significant.

Following these considerations, we have calculated the most-investigated example, namely, man, and we find actually a striking concordance between the *Abnützungsquote* determined in physiological experiment and as calculated from the growth curve. As mentioned above, the *Abnützungsquote* for man is to be assumed as about 45-75 g. protein for a man weighing 70 kg., corresponding to the physiological protein minimum of about 80 g., all energetic needs being supplied by non-protein nourishment. We have chosen the well-known growth data of Quetelet, calculating the growth constants for the last growth cycle, lasting from the 13th year to close of the progressive phase. As is to be seen (Table 10), for human growth $k = 0.1414$ per year, or $K =$

TABLE 10

Calculation of growth constants of man. Data of Quetelet for males

AGE IN YEARS	WEIGHT IN KG.	$\lambda = m^{\frac{1}{3}}$	$\frac{d\lambda}{dt}$	λ^2	$\lambda \cdot \frac{d\lambda}{dt}$
13	33.1	.....	.....	.....	.....
14	37.1	3.3352	0.1216	11.124	0.4056
15	41.2	3.4539	0.1161	11.929	0.4010
16	45.4	3.5674	0.1114	12.726	0.3974
17	49.7	3.6767	0.1049	13.518	0.3857
18	53.9	3.7772	0.0927	14.276	0.3502
19	57.6	3.8620	0.0634	14.915	0.2449
20	59.5	3.9040	0.0394	15.242	0.1538
21	61.2	3.9408	0.0364	15.530	0.1434
22	62.9	3.9769	0.0349	15.816	0.1388
23	64.5	.....	.....	.....	.....
9	....	33.4921	0.7208	125.067	2.6208

Normal equations:

$9E - 33.4921k = 0.7208$

$33.4921E - 125.067k = 2.6208$

$k_{year} = 0.1414$

$3k = 0.0011655$ per day. Multiplying by 70, as average weight, we obtain a daily protein loss of 0.0814 kg. or 81.4 g., corresponding to the *Abnützungsquote* and physiological protein minimum.

Of course, it is not at all to be assumed that constant K everywhere is to be identified with the protein *Abnützungsquote*. In the mathematical formulation, K represents some growth-inhibiting, mass-proportional factors in general. We have considered as yet the simplest assumption, namely, that these inhibiting factors are represented by the *Abnützungsquote*. But it is obvious that there are also other inhibiting, roughly mass-proportional factors, which, as yet quantitatively unanalyzable, are implied in K. First of all, in the organism there occur not only processes of disintegration whose end products are excreted, but also processes of transformation, in which substances originally participating in cell metabolism drop out of it (production of horn substances, protamins, histones, etc.). Cytologically speaking, there are (besides manifest cell destruction as given by the *Abnützungsquote*) also the processes of histological differentiation, leading to the formation of different metaplasms, elastic tissue, bones, cartilage, horn substances, pigments, nerve and muscle fibers, etc. Though these elements do not leave the body, they do more or less leave metabolism of building materials as shown by their lack of growth and regenerative capacity in the fully-developed state. Therefore, these elements must be entered as more or less stable products, on the debit side of metabolism of building materials. With this progressive differentiation the decrease of water content and increase of dry substance with increasing age is obviously connected. Equally it would be nonsensical to limit the needs of building material in the growing organism to protein requirement, omitting, for example, the requirements of calcium for skeleton development, etc. But in the first place the fact that the actual growth curves follow accurately the equations given above proves that actually growth is effectuated by the balance of furthering surface factors and inhibiting mass factors; and secondly, the fact, that the latter in a rather extended series of examples can be identified with physiological *Abnützungsquote*, proves that in these cases (cases of slow growth) the latter is the chief limiting factor or master reaction.

SUMMARY

1. Organic growth is considered as resulting from the counteracting of destruction and construction of building materials. It is assumed that

the former is a function of volume, the latter a function of surface. Morphological and physiological arguments in favor of these assumptions are given, and the equations for linear and weight growth following from them are derived.

2. Growth in the cyprinodont, *Lebistes reticulatus,* is analyzed, and it is shown that the whole curve of postembryonic growth can be calculated through the equations mentioned above.

3. In higher vertebrates the growth curve is subdivided in cycles caused by events leading to changes in total metabolism, such as birth, ablactation, hormonal activity of sexual glands, etc. These cycles manifest themselves in characteristic changes of growth rates and growth constants. With respect to these cycles, it is possible to calculate the very complicated growth curves of mammals with all detail by means of the growth equations here developed.

4. From the growth equations the numerical values of the metabolic constant K (i.e. destruction of building material per unit of weight and time = *Abnützungsquote,* Rubner) and H (i.e. construction per unit of surface and time) can be calculated. It has been shown in a foregoing investigation that the values of catabolic metabolism of building materials as calculated by the growth curves agree with the values determined in physiological experiment. In this way it is shown (1) that the said constants are not mathematically constructed entities, but physiological realities; and (2) the identity of constants found in two so different ways is a proof for the correctness of the theory.

5. As regards mammals, the different concepts concerning protein metabolism are analyzed, and it is demonstrated that probably protein loss in inanition gives the best available estimation of *Abnützungsquote.*

6. The identity of catabolic metabolism, as calculated from the growth curve, with experimentally determined *Abnützungsquote* and physiological protein minimum in man is demonstrated.

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MALTHUS' CONCEPTION OF THE CHECKS TO POPULATION

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I

THOUGH Malthus concerned himself with an imposing array of subjects in the field of population, he none the less acknowledged that his interests lay in a special direction. With respect to views known or current in his day, he made no pretense to originality. Thus the proposition that population must be kept down to the level of the means of subsistence, he styled as "an obvious truth taken notice of by many writers."[1] That poverty and misery arise from a too rapid increase of numbers, though vitally important in the fabric of his theory, he represented as "seen ... so long ago as the times of Plato and Aristotle."[2] The task he set for himself stretched beyond the bounds of a mere recapitulation or enlargement of widely-accepted ideas.

What then was Malthus' guiding purpose? The answer is to be found in his own observation: "The chief object of my work was to inquire what *effects* these laws, which I considered as established in the first six pages had produced, and were likely to produce on society..."[3] But the effects of the superior power of population, in so far as the leveling of population to means of subsistence was concerned, were the checks of vice, misery, and moral restrain. In point of fact, this bent of his thought was already revealed in the first edition of the *Essay on*

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[1] Preface to first edition of Malthus' Essay on the Principle of Population. (Royal Economic Society reprint, *London*, 1926), p. III.

[2] Preface to second edition of Malthus' Essay (*London*, 1803), p. IV.

[3] Third edition (*London*, 1806), II, Appendix, p. 520.

Note also "The Principal object of the present essay is to examine the effects of one great cause intimately united with the very nature of man..." (Second edition, pp. 1-2.) Malthus goes on to declare that these effects "have been almost totally overlooked."

Population where he indicated that he knew of no inquiry "into the means or modes" by which the leveling of population is effected;[4] i.e. into the workings of vice and misery, the two great depopulating causes envisaged in that period.

Though checks had been noted by other writers,[5] they had not as yet won a recognized place in social philosophy. The thought had not impressed itself on the minds of these writers, as on Malthus', that in the effects of the superior power of population is to be discovered a plausible explanation of much of the world's vice and misery, of a considerable proportion of economic inequality, and of the vanity of any faith in the "very great future improvement of society." The checks were thus sufficiently important to be given a prominent place in the field of population analysis. Here was a new subject calling for a detailed historical examination, for articulation with the existing body of social knowledge, and for a meaningful interpretation.

A brief exposition of the origin and character of the checks to population is found in the first two chapters of the several editions of Malthus' Essay. The treatment of the subject in the first edition shows Malthus moving forward boldly though not unsearchingly in an obscure region not yet traversed by beaten tracks. The terms "positive" and "preventive" do not appear until Chapter 4 is reached, and some additional, nearly forgotten, members of the family of checks do not appear until the end of Chapter 5. It is in the second chapter of the third edition that the system of checks appears in finished form.[6] This version may be tabulated as follows:

Ultimate check: due to a want of food "arising from the different ratios according to which population and food increase."[7]

[4] First edition, Preface, p. III.

[5] Michael T. Sadler, for example, refers to an anonymous work, the Philosophical Survey of the Animal, translated from the French and appearing after the middle of the eighteenth century, in which Malthus' list of positive checks, with the sole exception of "bad nursing" is enumerated. See the Law of Population (*London*, 1830) I, pp. 48-49.

[6] This chapter is reproduced without any change in phraseology in the seventh edition, the first to appear after Malthus' death.

[7] The term "ultimate" does not imply the potential food resources of the world. The ultimate check, Malthus states, is never the immediate one except in case of famine. The pressure upon food is not immediately responsible for checking the growth of population under ordinary circumstances. It is, however, the seedplot

Immediate check or checks:[8] the sum of all the preventive and positive checks. This totality is in turn resolvable, according to form, into vice, misery, and moral restraint.

I. Preventive checks:

A. Voluntary: peculiar to man on account of the superiority of his reasoning faculties.

1. Moral restraint: abstention from marriage until one has the requisite ability to support a family, with strict moral conduct during this period.
2. Prudential restraint: like moral restraint implies non-marriage for prudential reasons, but with the possibilty of a deviation from the path of moral conduct.
3. Preventive checks that come under the head of vice.

B. Involuntary: though spoken of in relation to plants and animals, their application to man is not excluded.

II. Positive checks: "every cause whether arising from vice or misery which in any degree contributes to shorten the natural duration of human life."

A. Unavoidable: checks which appear to arise unavoidably from the laws of nature.

B. Avoidable, or checks of a mixed nature, e.g. "wars, excesses, and many others which it would be in our power to avoid."[9]

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out of which the actual checks, both preventive and positive, grow. The want of food in Malthus' words "is the most efficient cause of the....immediate checks to population." (Third edition, II, p. 56.)

[8] The word "immediate" calls attention to the functioning of the checks as a totality in the present actual work of repressing population. The checks, writes Malthus, are "the immediate causes of the slow increase of population." (Third edition, II, p. 49.) Though the "immediate check" is not listed as a separate category in the second edition, the following comment appears: "The sum of all the positive and preventive checks taken together, forms undoubtedly the immediate cause which represses population but we can never expect to obtain and estimate accurately this sum in any country." (Second edition, p. 180.)

[9] Malthus contends that the general consequence of vice is misery. "The preventive checks that come under the head of vice" thus eventuate into the positive checks specified as avoidable, the one type merging into the other. The idea of a positive check we bring upon ourselves harmonizes with the greater moderation, exhibited from 1803 on, in the elucidation of this problem. The checks are now said to result "principally", not solely, from an insufficiency of subsistence, and the

III. Very powerful and obvious checks:[10] to account for a few instances where population fails to keep up to the level of the means of subsistence.

IV. Checks to the increase of plants and animals:

A. Positive.

B. Preventive—involuntary (first mentioned in the third edition. In the second Malthus argues that as plants and animals have apparently no doubts about their offspring the checks to their indefinite increase are all positive).

II

What is the function of the checks? By and large, the checks exist to repress the superior power of population or to keep population down to the level of the means of subsistence. This is explained by laws of nature, which on the one hand make food indispensable to life and on the other, impose limits on both nourishment and room. Another part of nature (permanent, indeed) is the passion between the sexes, which manifests itself through early marriage in a constant effort to an increase in numbers.[11]

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want of food is only the most efficient cause of their operation. (Second edition, p. 336.) Indeed, the notion of a positive check we bring upon ourselves is implicit in the very conception of moral restraint. The setting forth of moral restraint as "the strict line of duty," the doctrine that it is our offenses against the virtue of moral restraint that bring "the evils attendant upon vice, and the pains that accompany the various forms of premature death," and the recognition of the possibility of almost totally removing these evils, suggest that the misery induced by the disregard of moral restraint is in reality a positive check we bring upon ourselves. (See Second edition, pp. 504, 502, 505.)

[10] See third edition, I, p. 29; also Appendix, II, pp. 556-557.

[11] A union having taken place, procreation, in the absence of any depopulating causes, is taken for granted. The passion between the sexes, which he hopes will not lose it vividness, "prompts to increase." "When a man marries," Malthus says, "he cannot tell what number of children he shall have, and many have more than six." (Second edition, p. 595). So compelling is this tendency to increase that "it is found to act even in the most vicious societies." (*Ibid,* p. 12.)

At one point Malthus intimates recognition of a distinction between fecundity and passion between the sexes. He says: "The fecundity of the human species is, in some respects, a distinct consideration from the passion between the sexes, as it evidently depends more upon the power of women in bearing children than upon the strength and weakness of this passion." But fecundity, he observes, is a law

The checks are regulatory agents conditioning the existence of a growing, stationary, or declining population. Though visioned as elements of a natural order, they are responsive to shifts in such economic and institutional factors as commercial and agricultural systems, marriage, property ownership, forms of government, poor-laws, etc.[12] Malthus himself insisted that a clarification of the behavior of the checks would require a new approach to history, one evincing more interest in the state of the lower classes and promising "a clearer insight into the internal structure of human society."[13]

"The rates of increase," Malthus writes, "are fast or slow according as the pressures of these checks is light or heavy."[14] The checks, in other words, are "obstacles to the increase of population," or the causes of the slowing up of the process of growth. The pressure on the means of subsistence is thus reflected in the pressure of the checks on society. Under conditions of ample resources and wholesome morals this pressure may be so slight as to allow numbers to double in twenty-five years. Under less happy or markedly adverse circumstances, the period varies. Doubling may require some hundreds of years; or population may become stationary, or decline. On rare occasions the checks precipitate consequences so deleterious as to arrest population permanently at a level below that set by the means of subsistence. In all these cases they fill a rôle of fateful significance in human experience.

An examination of the various editions of the Essay shows Malthus developing his inquiries in sundry directions. He dilates on the wider aspects of the problem, i.e. the functions of the checks and their actual operation in various societies in all parts of the world and through several millenia of history; he comments on their interdependence, efficiency, intensity, and variations in pressure. He shows their relation to what he calls the retrograde and progressive movements of society and points to the possibility, when population is redundant, of some

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"similar in its great features to all the other laws of nature. It is strong and general, and apparently would not admit of any very considerable diminution, without being inadequate to its object." (Second edition, p. 491.)

[12] In this list should be included the "interrupting causes," factors like manufactures, wars, sickly seasons, poor laws, emigrations, etc., which interfere with the regularity of the oscillatory movements of population. Seventh edition. (Everyman's Library Reprint) I, p. 17.

[13] *Ibid*, pp. 16-17.

[14] Appendix to fifth edition (1817), published in seventh edition (*London*, 1872), p. 515.

depopulating causes superseding the necessity for others, i.e. of the substitution of one class of checks for another.

To remove the checks is impossible; but enlightened governments can make them operate more equably and, in a sense, maneuver them to produce the least evil.[15] Indeed, Malthus, actually grades them according to an ethical scale. "I have not the slightest hesitation in saying that the prudential check to marriage is better than premature mortality,"[16] he writes. Moral restraint is, in fact, "the strict line of duty," "the proper check to population"[17]—a salutary and practical bulwark against what William Hazlitt ironically dubbed "the untamed Beelzebub that was only kept chained down by vice and misery."[18]

In the light of his assumptions on population increase and subsistence, it is natural that Malthus should have placed great emphasis on the constant action of the checks. Hence such terminology as "constant check," the "constant effort of the checks," or checks "which are constantly operating with more or less force in every society."[19] This was a prolific source of misunderstanding and adverse criticism. The opposite view (already expressed by Condorcet before the appearance of the First Essay), that the evil results of excessive population were

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[15] See Malthus' Principles of Political Economy (*London*, 1820), pp. 250-252.

[16] Quoted in Cox, Problems of Population (*New York*, 1923), p. 202.

[17] Second edition, pp. 504, 494.

[18] Hazlitt, Political Essays (*London*, 1819), p. 423.

Malthus' endorsement of moral restraint is by no means a blanket approval of all species of preventive checks. Though Malthus was in a position to know of the existence and even progress of post-marital birth control, he seems to have felt a distaste for dealing openly with this subject. Whatever comments he made, relevant to it, betoken a determined hostility to any forms of restriction that seemed to him artificial, unnatural, or immoral. The following, in answer to the charge that he recommended Condorcet's check, (regarded by some as birth control) may serve as an illustration: "I have never adverted to the check suggested by Condorcet without the most marked disapprobation. Indeed I should always particularly reprobate any artificial and unnatural modes of checking population, both on account of their immorality and their tendency to remove a necessary stimulus to industry. If it were possible for each married couple to limit by a wish the number of their children, there is certainly every reason to fear that the indolence of the human race would be very greatly increased and that neither the population of individual countries nor of the whole earth would ever reach its natural and proper extent." (Appendix to fifth edition, *op. cit.*, pp. 511-512.) See also Norman E. Himes, "Note on Malthus' Attitude toward Birth Control," Appendix A, in Illustrations and Proofs of the Principle of Population, by Francis Place (Houghton Mifflin Co., reissue, 1930), pp. 283-290.

[19] Seventh edition, I, Chapter 2.

of no present concern though they might possibly have to be reckoned with in the future, was frequently adverted to by Malthus' critics. Thus Godwin insisted that there were no bounds to the physical power of the earth to supply subsistence, and Henry George that "the limit of subsistence in any particular place is not the physical limit of that place but the physical limit of the globe."[20] Malthus' position, however, was that potential resources do not alter the proportion between the natural increase of population and of food.

The point stressed by Michael T. Sadler and other critics that increasing numbers are generally conducive to the enjoyment of better living conditions and comfort was of course recognized by Malthus. He alludes also to the converse fact that a heavier pall of misery may lie on regions thinly inhabited.

His animus is not against "the absolute number of people," but against an unfavorable proportion between population and food. Hence the constant operation of the checks need not be regarded as inimical to a progressive advance in population. The checks "prevent population from outrunning the means of subsistence,"[21] but they do not prevent efforts to increase subsistence. Malthus' writings are replete with examples showing how both subsistence and population may increase advantageously.

It is perhaps not sufficiently appreciated that Malthus held fairly orthodox views on the matter of population growth. He complains with some feeling against critics who regard him as an enemy to population growth. Even in the First Essay, which represents him in his most truculent mood, he points out that "there is not a truer criterion of the happiness and innocence of a people than the rapidity of their increase."[22] What is necessary for a rapid increase is a great and continued demand, and a high real price for labor. The important consideration is the proper proportioning of population and food; this accomplished, population might continue to increase for ages.[23] He even emphasizes the thought that it is the object of the Creator to replenish

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[20] Henry George, Progress and Poverty (*New York*, 1932), p. 132.

[21] Seventh edition, I, p. 312.

[22] First edition, pp. 108, 137.

[23] Seventh edition, II, p. 174. Speaking of the assertions of some of his critics, he says, "they proceed upon the very strange supposition that the *ultimate* object of my work is to check population as if anything could be more desirable than the most rapid increase of population, unaccompanied by vice and misery." (Appendix to third edition, II, p. 515.)

the earth and, not without a shade of paradox, that the world would not have been peopled but for the superiority of the power of population. To justify this view he draws a distinction between partial or incidental evils, which human foresight can mitigate, and "the qualities of strength and generality" pertaining to laws of nature, necessary for the attainment of the great purpose of creation.[24]

Yet the logic of his argument underscored the need for regulation, since the means of adding to the food supply falls short of the power to increase numbers. Time and again he recognizes the possibility and evil consequences of a redundant population. A redundant population, he tells us, "is evinced by the inability of the poor to maintain all their children."[25] He speaks of circumstances that provide unnecessary children, of the excessively large families of the poor, of the duty of each individual not to marry until he has a prospect of supporting his children, and of the advantage of increasing numbers through a lessening of mortality as against a condition of high birth rates coupled with high death rates.[26] What he was really after is well brought out in his statement that the two grand desiderata are a great actual population and a state of society "in which abject poverty and dependence are comparatively but little known."[27] Even the intentions of the Creator are set forth as the replenishment of the earth with "a healthy, virtuous, and happy population."[28]

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[24] Seventh edition, II, pp. 157-158.

[25] *Ibid*, p. 223. Note also "it is of the very utmost importance to the happiness of mankind that population should not increase too fast." (p. 158.) But this is a concession to his feeling that it is "regulation and direction which are required with regard to the principle of population, not diminution or alteration." (p. 159.)

[26] *Ibid*, I, p. 290.

[27] *Ibid*, II, p. 172.

[28] Appendix to third edition, II, p. 509. These views, stressing "the desirableness of a great and efficient population" are somewhat suggestive of modern ideas of an optimum. But "efficient population" did not mean to Malthus (what it does to many exponents of an optimum) the most productive ratio between population and natural resources, and the highest per capita efficiency and income in terms of the current conception of diminishing returns. Malthus had in mind "that part of the population which is of an age to be employed effectually in agriculture, commerce, or war," an element particularly important because it was a principal factor in determining "the power of a country to increase its resources or defend its possessions." But though efficient population implied "constant employment in either agriculture or commerce," he visualized it as an aspect of the basic problem of the right ratio between population and food. (Appendix to third edition, II, pp. 510-511.) Nor would Malthus follow the protagonists of the optimum in

It will be recalled that, by the time of the appearance of the second edition, Malthus had espoused the opinion that the checks result *principally* (italics not in original) from insufficiency of subsistence or from want of room and food. The appelation "checks" simply brings to the fore the function of vice, misery and restraint as means or ministers of depopulation. By and large, then, the checks grow out of evil and, with the exception of moral restraint, proliferate new evil, though in the stretch of time they afford relief from redundancy and pave the way for improvement. But Malthus protests against the charge that he regards vice and misery as remedies. "Of course my ultimate object is to diminish vice and misery," he declares, "and any checks to population which may have been suggested are solely as means to accomplish this end."[29]

Already in the 1803 edition the argument is set forth that nature's habit of seizing upon our weakest parts is a discipline to teach us, "to make all parts strong and to chase vice and misery from the earth." But if this was his purpose, he no doubt opened himself up to sharp criticism by a tendency to indiscreetness of phrase and over-statement in dealing with this subject. It is not surprising under the circumstances that Hazlitt, for example, should have persisted in saying that the Malthusian theory considered vice and misery "as a salutary check" and that his doctrine led to the assumption "that we ought in mere kindness and compassion to give every sort of indirect and underhanded encouragement...to the providential checks of vice and misery." See The Spirit of the Age (Oxford University Press, *London*, 1928), pp. 151-152.

This part of the discussion opens up a thorny problem. The pressure of population is capable of producing untold evil, of drowning man in vice and misery. But it is also good, a method provided by the Creator

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sanctioning the aim of a "stringent restriction of the population growth" (as Professor A. B. Wolfe puts it) at some ideal point, not only because such policy would conflict with "the end of nature in the peopling of the earth," but also because it would obviously necessitate modern techniques of birth regulation.

Edwin Cannan observes, "Malthus...was so far infected with the prevalent opinions of his age, that the idea of there being too many people was quite strange to him." (Theories of Production and Distribution, second edition, *London*, 1903, p. 137.) This view is reëchoed by Professor F. R. Fairchild, who states that Malthus' emphasis on population has fortified the prevailing conception of "mere numbers as an end in itself." (Proceedings of the World Population Conference, *London*, 1927, p. 76.) That Malthus did not regard mere numbers as an end in itself, is shown by his emphasis on an efficient, healthy, virtuous, and happy population, his unstinted criticism of imprudent procreation, and his reiterated aversion to an unfavorable proportion between population and food.

[29] Appendix to third edition, II, pp. 515-516.

to overcome indolence, to stimulate the desire for food, to replenish the earth and to produce the activity "necessary to the improvement of the human faculties."[30] These are basic factors in the long-run process of society. Malthus succinctly puts the case in saying that "evil seems to be necessary to create mind."[31] Thus evil stems from "the general laws of nature." But the end of nature is identified with the wider purposes of creation. The laws of nature, says Malthus, are the laws of God.[32]

To escape this dilemma Malthus undertakes to distinguish between the consequences of the principle of population and the qualities of strength and generality characteristic of laws of nature and needed to carry out nature's purpose. The evils coupled with human fecundity are now described as partial or incidental to the "necessary qualities of strength and generality." But it is hard to see how this distinction solves the problem. If the incidental evils are part of the texture of the general laws of nature, so necessary for the achievement of great

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[30] Seventh edition, II, pp. 157-158. This aspect of Malthusian doctrine does not seem to have undergone any significant change in the entire series of editions of the Essay. It is elaborated at some length in Chapters XVIII and XIX of the first edition; the essential principles are restated in Book IV, Chapter 1, of the sixth and of the seventh editions. The dominant idea is expressed in this sentence: "The principle according to which population increases prevents the vices of mankind, or the accidents of nature, the partial evils arising from general laws from obstructing the high purposes of the Creator." (First edition, p. 365.) But this species of prevention is incarnate in the system of nature itself and pertains to the ostensible purposes of creation, in contrast to the conscious exercise by the individual of prudential or moral restraint to guard himself against misery or the loss of his comforts and conveniences.

Hence another function of the order of nature that generates the checks is to keep the desires alive and active. Malthus argues that these desires are so necessary to our being "that they could not be generally weakened or diminished without injuring our happiness." (Seventh edition, II, p. 153.) The desires for food, property, and marriage are in a healthier state when they encounter some obstacles. The sex passion, he observes, is stronger "and its general effects in producing gentleness, kindness, and suavity of manners much more powerful where obstacles are thrown in the way of very early and universal gratification." (*Ibid*, p. 156.) The desire for means of subsistence too would be "confined in its effects...were it not for the strong and universal effort of population to increase with greater rapidity than its supplies." (*Ibid.* p. 158.) On the one side this effort of population unleashes the checks; on the other, it wards off the vice of indolence and keeps the desires, without which the "improvement of human faculties" were impossible, fresh and undiminished.

[31] First edition, p. 360.

[32] Appendix to third edition, II, p. 527.

ends, how is one to know at what point the broad cosmic aspects of evil shade off into the partial type, i.e. the type that can be "greatly mitigated . . . by human energy and virtue."[33] Since moral restraint is admonished as "the strict line of duty," what criterion is there for knowing just how far interference with the seeming designs of nature may safely proceed? Where is the line to be drawn between the portion of evil necessary to incite exertion, to create mind, to spiritualize life, and the portion which may be dispensed with to the advantage of all concerned?

Related to this problem is a phenomenon of supreme importance in population theory, viz., that life may become so degraded as to nullify all hope or desire for improvement. "Even poverty itself," Malthus asserts, "which appears to be the great spur to industry, when it has once passed certain limits, almost ceases to operate. The indigence which is hopeless destroys all vigorous exertion and confines the efforts to what is sufficient for bare existence. It is the hope of bettering our condition, and the fear of want, rather than want itself, that is the best stimulus to industry, and its most constant and best directed efforts will almost invariably be found among a class of people above the class of the wretchedly poor."[34] Thus, the "indolence of man," unencumbered by the stern discipline of the law of population, reestablishes its dominion in the element that is sunk in hopeless indigence, nullifying the meliorative action of the laws of nature and enmeshing society in its folds.[35] Under circumstances involving such variations in the possible results of the processes of nature as human striving energized by want, or specially stimulated by the hope of a better standard of living, or rotting away in a quagmire of drab, incurable indigence, the trail of such

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[33] Second edition, II, p. 157. An illustration is found in Galton's criticism of Malthus' "prudential delay in the time of marriage" because of its effects on the race. Galton writes: "The doctrine would only be followed by the prudent and self-denying; it would be neglected by the impulsive and self-seeking... There would have been a little relief during one or two generations but no permanent increase of the general happiness, while the race of the nation would have deteriorated. The practical application of the doctrine of deferred marriage would therefore lead indirectly to some mischievous results that were overlooked owing to the neglect of considerations bearing on the race." (See Inquiries into Human Faculty and its Development, Everyman's Library, p. 207.)

[34] Second edition, p. 475.

[35] The preventive check or more particularly the checks to increase "from reason and foresight" may be destroyed by ignorance and despotism, by a "system of equality and community of goods," or by poor-laws. (See seventh edition, II, 143, 27, 208.)

meliorative action becomes almost unidentifiable. If the exercise of the moral check is justifiable not only in efforts to escape indigence, but in maneuvers to better one's condition, the task of steering a path through a labyrinth of utilitarian ends intertwined with the ultimate ends of creation becomes an almost impossible one.

The evils associated either with the origin or operation of the checks do not affect equally all classes of society. The preventive check operates in all ranks, but it is the lower classes that need it most.[36] The positive check "is confined chiefly, though not perhaps solely, to the lowest orders of society."[37] This aspect of Malthusian doctrine became the subject of hot dispute. It drew forth caustic comments from Hazlitt, and roused the ire of Sadler. Macaulay, who took up the cudgels for Malthus asked, "Why is it more inconsistent with the divine goodness that poor men should suffer an evil from which rich men are exempt, than that a particular portion of the community should inherit gout, scrofula, insanity and other maladies?"[38]

But if economic inequalities are vindicated by the laws of nature, it is principally because of the manner in which the action of population nullifies any grandiose "systems of equality." It was this phase of the subject that led Malthus to an analysis that has left a deep mark on the literature of socialism.[39] Using the principle of population as a criterion, he points out that if property were divided equally there would be no means of curbing the power of population. "The encouragement and motive to moral restraint," he declares, "are at once destroyed in a system of equality and community of goods." Encouraged by general

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[36] The Malthusian point of view is markedly different from that of modern eugenics. Malthus was alarmed by the increase of vice and misery and the evils of redundancy—not by germinal inferiority or a deterioration of the race. The terms used by Karl Pearson at the beginning of this century, viz., "anti-social stocks," "degenerate parents," "the unfit parent," (see "The Problem of Practical Eugenics" in James Ford's Social Problems and Social Policy, pp. 328-332) find no place in Malthusian literature.

[37] First edition, p. 71.

[38] T. B. Macaulay, "Sadler's Refutation Refuted" in Historical and Miscellaneous Essays (*New York*, Sheldon & Company, 1862), II, p. 276.

[39] Though Hazlitt charged that the argument was a restatement of views from Wallace's Various Prospects of Mankind, Nature and Providence, Malthus himself traces it back to Aristotle. He writes "It appears...that Aristotle clearly saw that the strong tendency of the human race to increase, unless checked by strict and positive laws, was absolutely fatal to every system founded on equality of property." (Second edition, p. 168.)

benevolence, numbers would increase "faster than in any society that has ever yet been known," and the whole of society rather than a mere part, would find itself weighed down by want and misery.[40] Ruling out artificial regulations of population as unnatural and cruel, the only alternative would be the reestablishment of private property and of responsibility to support one's own children.[41] Since individual poverty can be allayed by thrift and postponement of marriage, the man who fails to practice these virtues has only himself to blame.

III

By and large, the preventive check implies abstention from early marriage; among the lower classes on account of want or the fear of want, and among the higher classes on account of lowering one's rank in life. The lessening of the proportion of births consequent upon the operation of this check, whether reconcilable with moral principles or not, was according to the Malthusian point of view a matter of prolificness rather than modification in bodily structure. Malthus, in contradistinction to Herbert Spencer, who enunciated the thesis that human fecundity diminishes with the progress of civilization, does not regard organic change as a decisive factor in the regulation of numbers.

But he does not confine himself to this limited conception of the problem. The preventive check also arises from a large proportion of the population above puberty dying unmarried. This, in turn, means

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[40] Seventh edition, II, pp. 27, 14, 18.

[41] Described as "a refinement of absurdity" by Hazlitt, the Malthusian argument reappears in the works of such writers as John Stuart Mill, J. B. Clark, and F. W. Taussig. See J. S. Mill, Principles of Political Economy (*New York*, D. Appleton and Company, 1872, from the fifth London edition.) I, pp. 245-246; J. B. Clark, "Education and the Socialist Movement" in Representative Essays in Modern Thought (Steeves and Ristine, 1913), pp. 464-465; F. W. Taussig, Principles of Economics, third revised edition, II, (1923), pp. 246-247.

The argument has of course been met by writers of the Socialistic School—See William Thompson, An Inquiry into the Principles of the Distribution of Wealth Most Conducive to Human Happiness (*London*, 1824), pp. 545-562; "Population and Over Population" in August Bebel's Woman under Socialism (Labor News Company, *New York*, 1904), pp. 354-371; Bernard Shaw, The Intelligent Woman's Guide to Socialism and Capitalism (*New York*, 1928), pp. 83-92. On the Marxian reaction to the Malthusian treatment of the population problem see S. M. Levin, Marx *versus* Malthus, Papers of the *Michigan Academy of Science, Arts, and Letters*, XXII (1936), pp. 243-258.

that prevention may be merely a result of the premature death of potentially marriageable individuals, i.e. of individuals capable of reproducing themselves. But all forms of premature death are linked to the positive check. The latter in some of its forms thus takes on the guise of a double-edged check, being both positive and preventive. Again, the checks taken *en masse* may be considered as preventive in the sense that they "prevent population from outrunning the means of subsistence," or, if very powerful, even from reaching that level.

There is thus no clear line of demarcation between the positive and preventive checks. Technically the preventive checks prevent a new population from being brought into the world, but they may also, through vice and misery, cut down an existing one. Similarly, misery proper may induce recourse to preventive arts that come under the head of vice,[42] or lead to the same result as prevention does by augmenting the mortality of individuals, otherwise destined to provide new parental stock.

From the outset, Malthus applied the principle of population to all "animated life," speaking of the checks to the increase of plants and animals as positive. In the third edition he enunciates a new viewpoint in these words: "The checks to the indefinite increase of plants and irrational animals are all either positive, or, if preventive, involuntary," an admission that some kind of preventive check applicable to plants and animals is at least possible. The distinction is also brought out in the statement: "The preventive check, as far as it is *voluntary*, is peculiar to man."[43] Though a voluntary preventive check for plants and animals

[42] See J. A. Field, Essays on Population (*Chicago*, 1931), p. 26. Vice is really of secondary importance. Malthus admits that the misery arising from want of room and food is "a much more powerful cause even than vice" in regulating numbers. (First edition, p. 109; seventh edition, I, p. 153.) Since "the general consequence of vice is misery," it is the latter that plays the leading rôle.

[43] Third edition, I, p. 16. It was left to Darwin to make a far-reaching application of this aspect of Malthusian theory. Darwin's reaction is brought out in the following passage: "As more individuals are produced than can possibly survive, there must in every case be a struggle for existence; either one individual with another of the same species, or with individuals of distinct species, or with the physical conditions of life. It is the doctrine of Malthus applied with manifold force to the whole animal and vegetable kingdoms, for in this case there can be no artificial increase of food, and no prudential restraint from marriage." (Darwin, *op. cit.* I, p. 79.) Malthus used the term "struggle for existence" and also "perpetual struggle for room and food," in his first edition. (See pp. 47-48.) Though he did not invent the term "survival of the fittest," he came very close to this conception. See first edition, p. 42; second edition, pp. 28-29.

is inconceivable, there is no necessary exclusion of the possibility of an involuntary preventive check applicable to organic life in general.

With regard to Malthus' new check of "moral restraint" and his stressing of the consequent difference in principle between the first and second editions, Professor Fetter writes: "A glance...at the first edition shows that in it moral restraint was distinctly recognized by Malthus as a check, in fact, was *greatly emphasized,* (italics not in original) though he did not call it by exactly that name."[44] That conclusion pivots on the fact that Malthus wrote "restraint almost necessarily, though not absolutely so, produces vice."[45] Restraint that does not produce vice is to all intents and purposes moral restraint. Pursuant to the same line of thought Malthus avowed that vice is only "a highly probable consequence" of the constantly operating check on population, and that "the ordeal of virtue is to resist all temptation to evil."[46] Field states,..."It is tolerably plain that the term preventive check was used in the original edition of 1798 in the sense of prudential restraint with an implied moral character."[47]

At best, a careful reader can discern only a hint of moral restraint in the First Essay; there is no sign of emphasis. The bias of Malthus' thought is evidenced in the opinion that "the slightest check to marriage, from a prospect of the difficulty of maintaining a family," may be regarded as misery.[48] It is in the edition of 1803 that the conclusion is reached that the exercise of the virtue of moral restraint to a certain degree "would...rather tend to increase than diminish individual happiness."[49] Certainly the earlier edition does not descant on the practicability and virtue of moral restraint nor offer a persuasive plea for its use by the laboring classes as their path of salvation.

Malthus' own estimate of the practical importance of the various branches of the preventive check are of further interest in this regard. He writes: "That branch of the preventive check which I have denominated moral restraint, though it has certainly had some share in repressing the natural power of population, yet taken in its strict sense, it must be allowed to have operated feebly, compared with the others." He

[44] F. A. Fetter, The Essay of Malthus, Yale Review, VII, (1898), p. 161.
[45] First edition, pp. 28-29.
[46] *Ibid.*, pp. 15-16.
[47] Field, *op. cit.*, p. 36. Field suggests that very probably it was Goodwin "who impressed Malthus with the inadequacy of his earlier opinions." (*Ibid.* p. 37.)
[48] First edition, p. 108.
[49] Second edition, p. 502.

confesses with admirable ingenuousness that the chief reason why he allowed himself to suppose the universal prevalence of moral restraint "was...to remove any imputation on the goodness of Deity," and even points to the fact that he has been accused of not allowing enough weight to moral restraint as a preventive measure. He concludes his lengthy survey of the checks with the acknowledgement that moral restraint "does not at present prevail much among the male part of society." It is only in modern Europe that "a delay of the marriage union from prudential considerations without reference to consequences ...may be considered...as the most powerful of the checks."[50] These are interesting admissions, in the light of his devotion to the cause of moral restraint.

IV

A distinction of considerable importance between Malthus' earlier and later opinion is found in the terminology of the second and third propositions on population. In the First Essay, Malthus maintained that population invariably increases when the means of subsistence increase. Subsequently he modified this to read "Population invariably increases when the means of subsistence increase, unless prevented by some *very powerful* and obvious checks."[51] This is a recognition that "there are very few instances, such as the negroes in the West Indies, and one or two others, where population does not keep up to the level of the means of subsistence."[52] In other words, Malthus, was obliged

[50] Citations from seventh edition, I, 153; II, 168; I, 315.

The preventive check as a prudentially motivated measure is particularly applicable to civilized society. Originally Malthus seems to have held that "vice was out of the question among savages," misery "with few local and temporary exceptions" being the only check. (See First edition, p. 44.) The preventive check was really inoperative. He remarks with regard to prudential considerations: "These considerations are calculated to prevent, and certainly do prevent, a very great number in all *civilized* nations from pursuing the dictate of nature in an early attachment to one woman." (First edition, p. 28.) In the second and later editions, however, Malthus declares that in savage life, when population has increased nearly to the utmost limits of food, *all the preventive and positive* checks will operate with increased force. He cites as examples "vicious habits with respect to...sex" and a number of positive checks, but no prudential or moral restraint. (Second edition, p. 15; seventh edition, I, p. 18.)

[51] Second edition, p. 16.

[52] Third edition, I, p. 29. See also footnote 2, seventh edition, I, pp. 18-19. In view of the strength of his feelings on this subject, it is not surprising that he

to admit "that the immediate checks to population might be too powerful to be overcome even by an abundance of food."[53] This he said is possible, "and foreseeing this possibility, I provided for it in the terms in which the second proposition of the Essay was enunciated."[54] His explanation is generally in terms of aggravated vice or misery, or the immediate checks to population operating with "excessive and unusual force."[55]

It was a delicate point. Malthus seems loath to admit that a population can be kept permanently below its natural level, except in extreme cases or under conditions of extreme violence. Such an admission would be tantamount to a denial of the tendency of population to outrun subsistence, the "great cause" constantly and powerfully operating since the commencement of society. It would pull the props from under the whole theory and make havoc of all the conclusions reached. Thus Malthus insists that "the constant effort towards population" is found to act even in "the most vicious societies"; even where population is stationary or retrograde, the cause he says cannot be traced "to a decay of the passions between the sexes," but to the operation of the checks. If in its oscillatory movement, population is retarded or sunk below the level of food, the situation is temporary; a return of plenty will again produce an increase. So prolific of good are the human passions, i.e. the sex passions and the desire for food and property, that Malthus disavows any thought of wishing them weakened. "Few or none of them," he remarks, "will admit of being greatly diminished without narrowing the sources of good, more powerfully than the sources of evil."[56]

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allowed himself to slip into the statement that population "with the sole exception" of the Negroes of the West Indies had been able to keep itself up to the level of subsistence. (Appendix to third edition, II, p. 558.) In the same appendix printed in the seventh edition (London, 1872) this is revised to read "almost with the sole exception above alluded to." (See p. 509.)

[53] Appendix to fifth edition, *op. cit.*, p. 517.

[54] *Ibid.* It may be pointed out that this also led to a restating of the third proposition in the words: "*These checks,* and the checks which repress the superior power of population...are all resolvable into moral restraint, vice and misery." "These checks," a term which does not appear in the first edition, obviously refers to the checks operating with great force. But Malthus continued to affirm that the second and third propositions are generally applicable in their unrevised form. See footnote 2, seventh edition, I, pp. 18-19.

[55] Appendix to third edition, II, p. 556.

[56] Citations from seventh edition, I, 15; first edition, p. 62; seventh edition, II, p. 156.

A certain ambiguity of treatment arises at this point. The checks operating with increased force may account for some forms of depopulation, but this does not satisfactorily account for the seeming breakdown of the "great cause," for the falling into decay of the tendency to increase, itself, for the failure of this tendency to operate even when food is plentiful.

Malthus had actually given himself a loophole, inasmuch as he had allowed that the checks were not solely due to want of food. Perhaps more emphasis should have been placed on a phenomenon that he himself recognized, i.e. the weakening of the desires on account of overindulgence. It is this problem that is involved in John Rae's criticism. Rae, who was acquainted with conditions in the Hawaiian Islands, was impressed by the fact that in these islands, despite plentiful resources and easy acquirement of subsistence, the native population, instead of increasing, rapidly diminished. Vice was not a check to the pressure of a *growing population*. The "Malthusian philosophy of population," he concluded, "accounts for the vital phenomena of healthy societies only, not at all for that of sick societies..."[57]

Noting what Malthus wrote about Greek and Roman slaves, West Indian Negroes, and English servants, conjoined with his rephrasing of the second and third propositions on population, it is impossible to accept the opinion that he concerned himself with the vital phenomena of healthy societies only. He surely realized that a given population may, for some reasons, fail to respond normally to the stimulus of an increased food supply. If any criticism is in point, it is one which may be more appropriately directed against the ineptness of his explanation of this type of experience.

V

It is interesting to note that, as the Malthusian inquiry widened its front, it encountered at various points, salient aspects of the problem of standard of living.[58] Habits of living apply in a direct manner to the motivation of the preventive check. They mingle with the very

[57] John Rae, The Sociological Theory of Capital (*New York*, 1905), p. 355.

[58] Malthus uses such terms as habits of living, standards of comfort, a taste for the decencies and comforts of life, and oddly enough, standard of wretchedness. "In most countries, among the lower classes of people," he observes, "there appears to be something like a standard of wretchedness, a point below which they will not continue to marry and propagate their species." (Seventh edition, II, p. 214.)

essence of a theory that posits the dependence of population on subsistence. Though Malthus recognized that "the comforts of the lower classes of society do not depend solely upon food,"...and that "they cannot be considered as in good state unless they have the command of some conveniences and even luxuries,"[59] he none the less appreciated that subsistence is conditioned by acquired habits and tastes. He saw that a country's supply of food can be made to support a greater population, if laborers can be forced to shift from a higher standard of consumption to a lower; on the other hand that the "difficulty of changing habits" may retard growth even when food is abundant.[60]

Malthus' conception of standard of living was in the main negative. It was certainly of less importance than the two ratios as a determinant of population growth. A wholesome standard operated primarily to induce people for a time to avoid marriage because of the fear of lowering their station in life, of being deprived of comforts and conveniences, or of having to engage in harder labor for a livelihood. It was an affair, primarily, of a reasoned calculation of relative advantages, at the point where a choice between marriage and competing wants had to be faced, rather than of the complex of feeling, sentiments, economic ambitions, and cultural pressures affecting the whole of life.

The circumscribed range of Malthus' thought on this subject is shown by the fanciful notion that the laborer could, by saving and wages, provide for the needs of a wife and five or six children without assistance. "Undoubtedly such a degree of prudential restraint," he says, "would produce a very striking melioration in the condition of the lower classes of people."[61] But could it, in respect to the post-marital period,

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[59] *Ibid.*, p. 130.

[60] Footnote, seventh edition (*London*, 1872), pp. 86-87.

[61] Seventh edition (Everyman's Library), II, p. 254. It is to be noted that Malthus envisaged the possibility of permanent good inuring to society as a result of an improvement in the habits of the people. Under one set of conditions the number of marriages would remain "nearly the same," but the death rate would fall, more children survive, and an added population be supplied in consequence of "a diminished mortality." If, on the other hand, the population had been long stationary, a change in habits might result in lessening the number of marriages and the number of children "lost in infancy from the diseases consequent upon poverty." (*Ibid.*, I, pp. 194-195.) Under these circumstances population would be kept at its level by a smaller number of births. Another possibility for laborers with large families, in individual instances, (which Malthus seems to construe as an advantage) is that these families can be taken care of by the sacrifice of "accustomed conveniences and comforts." (*Ibid.*, II, p. 135.)

even under the most sanguine expectations, be construed as the desired path for a progressive widening of standards? There is nothing in his pronouncements to show that he was ready to acknowledge the inhibiting influence of a rising post-marital standard as a tactic of defense against a surcharge of births. On the contrary, despite a hardened antipathy to public benevolence, he endorsed allowances to every child above six.[62]

But the fact is unmistakable that within the scope of his own system of ideas, Malthus shows a clear comprehension of the bearing of standard of living on the population problem. His emphasis on the relationship between standards of living and the preventive check, his observations on the circumstances which contribute to raise or lower standards,[63] and his recognition of their importance as a means of bettering the circumstances of the lower classes, are anticipatory of the wider views of a later period. Almost in the manner of contemporary writers, he cites examples of increased real wages, not occasioning an augmentation of marriages and births, but producing a marked improvement in the quality of goods consumed and an enhancement of the people's comforts and conveniences.[64] And finally he stresses the desirability of a "diffusion of luxury among the mass of the people." It is such diffusion, he declares, "and not an excess of it in a few, that seems to be most advantageous, both with regard to national wealth and national happiness."[65]

The shaping of Malthusian theory in this mold helps to clarify the question of the efficiency or inefficiency of the population checks. It is true that Malthus himself seemed to deny the possibility of the natural checks being inefficient.[66] From an ultimate standpoint, it is of course impossible for population to exceed the minimum food necessary for the continuance of human life. But this recognition of the ultimately effectual character of the natural checks is distinguishable from Cannan's view, that the efficiency of the checks is tested by the mere fact that

[62] *Ibid.*, p. 255.

[63] *Ibid.*, pp. 214-215.

[64] Malthus, Principles of Political Economy, pp. 253-254.

[65] Seventh edition, II, p. 253.

[66] Cannan quotes Malthus, as follows: "For undoubtedly as long as this continues to be a law of nature (that man cannot live without food), what are here called the natural checks cannot possibly fail of being effectual." *Op. cit.*, p. 137. Malthus' preoccupation with the intricate windings of this subject is shown by his expressing a special interest "to know the exact share of the full power of increase which each existing check prevents," in other words, the efficiency of each separate check. He notes, however, that he sees "no mode of obtaining such information." (Third edition, II, Appendix, p. 520.)

there are too many people, in other words, by the failure of the checks to be "as strong as it is *desirable* they should have been."[67] The numerous references to redundancy of population contradict Cannan's statement that the idea of there being too many people was quite strange to Malthus. Certainly, by the test of desirableness, the preventive check is inefficient in states of misery which defeat reason and foresight; so, too, are the checks *en bloc* when population increases "faster than the accomodations of a country with respect to habitations and food will allow,"[68] resulting in bad housing conditions, insufficient food, and disease. Using the same touchstone, the checks, in truth, may be deemed inefficient under circumstances of decadence as well as redundancy, for there is obvious inefficiency in the operation of the very powerful checks that defeat the purpose of a proper proportioning of population and food, and hinder the full cultivation of the earth.

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[67] Cannan, *op. cit.*, p. 137.

[68] Second edition, p. 342. The conceivable inefficiency of single checks is brought out in the explanation of the impossibility of determining the sum of the checks in any one country. "The excess of one check," Malthus writes, "is balanced by the defect of some other." (Second edition, p. 180.)

THE MASCULINITY OF STILLBIRTHS AND ABORTIONS IN RELATION TO THE DURATION OF UTEROGESTATION AND TO THE STATED CAUSES OF FOETAL MORTALITY[1]

BY ANTONIO CIOCCO

PROBLEM AND MATERIAL

IT HAS long been established that there are from 1250 to 1350 male stillbirths for every 1000 females born dead, and that the preponderance of male stillbirths is greatest in the early months of uterogestation and decreases with duration of pregnancy. A number of theories have been advanced to explain the apparent fragility of the male. Thus, Lenz (1931) sees in the unequal proportion of males and females among the stillbirths and abortions evidence of the action of a lethal gene. In contrast, Riddle (1927) regards the phenomenon as being a consequence of maladaptation of the male embryo to the female environment. Then there are others, such as Pfaundler (1936), who regard the preponderant masculine antenatal mortality as the effect of both genetic and environmental factors. All of these and other theories, when they do not draw analogies — at times unwarranted — from experimental studies in animal biology, are essentially the product of speculative rationalization based only on the two series of facts mentioned.

Since a clearer understanding of the mechanism of sex differences in intrauterine mortality in man has important bearings both on the problem of sex determination and on the causative factors of reproductive wastage, it would seem wiser to replace some of the efforts toward theorizing with a more intensive study of the causes of foetal mortality. With this in mind it has appeared desirable to initiate an analysis of the official vital statistics which present for a selected area of the United States—viz: Connecticut, Illinois, New Jersey, New York, Oregon,

[1] From the Department of Biology of the School of Hygiene and Public Health of the Johns Hopkins University, Baltimore, Maryland.

Washington, West Virginia, the District of Columbia, and Baltimore, Md.—data on the number of stillbirths classified according to sex, cause of death, and duration of uterogestation. Although more comprehensive information is contained in these data than is generally available for any sufficiently large sample of the general population, the material is defective in a number of ways. Aside from the gross error which necessarily accompanies any attempt to obtain detailed information regarding reproductive wastage, the official data can be criticized especially because all the states included in the area do not adopt the same definition of stillbirth and because the classification of causes of death manifest the usual confusion which results from the use of both descriptive and etiological terminology.

Nevertheless these data appear adequate enough to permit a preliminary inquiry regarding the sex differences in foetal mortality and in this paper a report is made of a study which has for its main objective to determine whether the masculinity of stillbirths varies in relation to cause of death and whether this variation is affected by, or itself affects, the well-known changes in the sex ratio of stillborn at different periods of uterogestation.

MASCULINITY AND DURATION OF UTEROGESTATION

During the years 1925 to 1934, there were more than 180,000 stillbirths and abortions of recognized sex registered in the selected area of the United States. Of these 57.1 percent were males, giving a sex ratio of 1330.3. When classified according to the duration of uterogestation, the distribution of the sex ratio and masculinity rate[2] is as in Table 1.

The proportion of males decreases rapidly from the second month to the fifth, then the decline is gradual until a minimum is reached at 7 months. Thereafter there is a slight increase. The monthly variation demonstrated in this table is apparently not a random fluctuation of the sex ratio for the total because the Lexis coefficient indicates that the standard deviation of the monthly fluctuations of the masculinity rates

[2] The sex ratio (number of males per 1000 females) has been included in the tabulations in homage to a time-honored custom. Actually the statistical analysis has been based on the masculinity rate $= \frac{100 \text{ males}}{\text{males} + \text{females}}$ which seems to me preferable from a number of standpoints.

TABLE 1

Masculinity rate and sex ratio of stillbirths that were registered between 1925 and 1934 in a selected area of the United States. Classified according to period of uterogestation

PERIOD OF UTEROGESTATION *(in months)*	MASCULINITY RATE	SEX RATIO	SEX RECOGNIZED IN *Number*	*Percent*
Under 2	69.5	2280.0	82	10.1
2	81.2	4311.3	563	27.8
3	78.3	3610.0	2388	63.2
4	66.8	2012.2	6401	89.5
5	58.3	1396.1	12541	97.4
6	55.1	1227.4	17857	99.0
7	52.7	1124.0	23109	99.4
8	55.5	1247.5	28903	99.6
9	57.4	1346.9	68932	99.8
10 and over	57.1	1332.8	2671	99.6
All stillbirths including those of unknown period	57.1	1330.3	181548	96.9

is more than ten times the standard deviation of the masculinity rate of the total.

The trend of the sex ratio by month of uterogestation as observed here is in good agreement with that reported by Auerbach (1912) on stillbirths and abortions in Budapest, and by Tschuprow (1915) on those of Budapest, Vienna and Paris. Schultz (1921), who has discussed this question in detail, believes that the high masculinity noted in the early months of uterogestation as deduced from official vital statistics is probably exaggerated due to error in recognizing the sex of the foetus in the early stages of intrauterine life. In his collection of over 1400 foetuses deriving in the majority from spontaneous abortions he found the sex ratio equal to 1210 in the third month, 1175 in the fourth, 1096 in the fifth, 875 in the sixth, 1085 in the seventh, 1333 in the eighth and 1676 in the ninth and tenth months. For the months preceding the seventh, these figures are markedly below those shown in Table 1. A glance at that table, which also gives for each month the

percentage of stillbirths in which sex was recognized, clearly indicates the difficulty encountered in sexing the individual and emphasizes, in agreement with Schultz, that the sex ratio in the early months of uterogestation is open to doubt. However, it is the opinion of most students, including Schultz, that among the antenatal deaths masculinity is at a minimum from the 5th to the 7th month and higher in the preceding and subsequent periods. A dissenter from the consensus of opinion is Boldrini (1930), who notes that among the very young embryos collected in the Carnegie Institution and described by Streeter (1920) and by Spaulding (1921), there are more females than males. Assuming that the collection of these two observers is a random sample of abortions he concludes that actually more females than males die in the very early periods of pregnancy, and that the difference is such that it more than balances the subsequent relative higher masculine mortality. Or, in other words, Boldrini accepts the concept of modern genetics that the probability of conception is equal for males and females, and believes that the preponderance of males at birth is a consequence of the relatively high female mortality which takes place in the first two months of pregnancy and which is never balanced by the later excess of deaths among the males. Boldrini's tentative conclusion is considerably weakened by the fact that a study by Wilson (1926), also on material collected in the Carnegie Institution, reveals that histological examination of the embryo is often in disagreement with the diagnosis of sex made by external examination. A considerable margin of error in the sexing is noted especially for embryos already labelled females and which his observations revealed to be males. In view of this, the basic evidence from which Boldrini draws his conclusions is not absolutely reliable. However, as has been pointed out (Ciocco, 1938) some such conclusion is necessary in order to explain the sex ratio if it is true that the same number of males and females are conceived. No other facts have ever been presented to substantiate a theory based on a preponderant female mortality in the early period of uterogestation and even if it is assumed that the official vital statistics of intrauterine deaths up to the fourth month of uterogestation are grossly erroneous and unreliable relative to sex, there seems to be no doubt that from the fifth to the seventh month there is a decline in the masculinity rate and that the monthly fluctuations of the masculinity rates from the fifth to the tenth month are greater than would be expected if chance alone were operating. All of this expresses the fact that the pattern of the duration of intrauterine existence is different among stillbirths of the two sexes.

This can be clearly seen from Table 2, which presents the percentage distribution of the dead expelled foetuses of each sex in relation to the duration of uterogestation.

TABLE 2

Percentage of recorded male and female stillbirths which have occurred at each month of uterogestation. Stillbirths registered from 1925 to 1934 in selected area of the United States

DURATION OF UTEROGESTATION *(in months)*	PERCENT STILLBIRTHS AT EACH MONTH	
	Males	Females
Under 2	0.06	0.04
2	.49	.15
3	2.01	.74
4	4.59	3.03
5	7.85	7.45
6	10.57	11.41
7	13.08	15.42
8	17.23	18.31
9	42.49	41.82
10 and over	1.64	1.63

Among the males the percentage of stillbirths which occur before the sixth month is higher than that found for the females in the same interval. The same is to be observed for the stillbirths which occur at the ninth month and after. On the other hand the proportion of females that have been born dead from the sixth to the eighth month is higher than that of males for the corresponding period. It is only too obvious that the extent of the differences between the two distributions determines the monthly fluctuations of the sex ratio, since if they were equal the sex ratio would be constant in all periods.

Of the factors that contribute to a different behavior of the two sexes with respect both to the frequency of intrauterine mortality and its pattern relative to duration of uterogestation, those included in the list of the causes of stillbirth are the ones which to a certain degree should throw some light on the significance of these observations. If medical knowledge were sufficiently advanced to describe precisely the etiological mechanism involved in each cause of stillbirth, the problem would be simply solved. As it is, the expectations must be limited.

MASCULINITY AND THE CAUSES OF STILLBIRTHS

In Table 3 are presented the distributions of male and female foetal deaths classified according to the major categories of the causes reported in the official vital statistics.

TABLE 3

Frequency distribution of the major categories of causes of male and female stillbirths in selected area of the United States from 1925 to 1934

CATEGORY OF CAUSES OF STILLBIRTHS	MALES		FEMALES	
	Number	*Percent*	*Number*	*Percent*
Causes not specified or unknown	25,993	25.08	19,309	24.79
Diseases of the placenta and membranes	10,884	10.50	8,203	10.53
Difficult labor	10,521	10.15	6,252	8.03
Prolapse and compression of cord	10,391	10.03	7,464	9.58
Abortion, miscarriage, premature birth	7,901	7.62	5,395	6.98
Albuminuria and other diseases incident to pregnancy	6,335	6.11	5,386	6.91
Malpresentation	5,323	5.14	3,713	4.77
Malformations	4,411	4.26	5,890	7.56
Asphyxia of child (cause not stated)	4,289	4.14	3,122	4.01
Death in utero	4,189	4.04	3,211	4.12
General diseases	3,816	3.68	2,879	3.70
Traumatism and overwork	3,157	3.05	2,138	2.74
Other specified causes	2,549	2.46	1,967	2.52
Other diseases or conditions of mother	2,325	2.24	1,752	2.25
Hydramnios	570	.55	473	.61
Hemorrhage of uterus	260	.25	186	.24
Inertia of uterus	198	.19	152	.20
Thrombosis of cord	171	.17	160	.21
Rupture of uterus	137	.13	104	.13
Retroversion of uterus	54	.05	22	.03
Predisposition of mother to abortion	50	.05	43	.06
Extra-uterine pregnancy	45	.04	41	.05
Antepartum hemorrhage	39	.04	30	.04
Prolapse of uterus	34	.03	14	.02
ALL CAUSES	103,642	100.00	77,906	100.03

For fully 25 percent of the stillbirths of both sexes the cause was not specified. Of the specific causes, Diseases of the placenta and membranes are the most important for both the males and females; separation of the placenta furnishes 53.6 percent of the males and 54.6 percent of the females classified under that heading. Next in order of importance among the major categories of death causes are Difficult labor, and Prolapse and compression of cord. Altogether these three categories account for 30 percent of the male deaths and a slightly inferior pro-

TABLE 4

Masculinity rate and sex ratio of stillbirths according to major categories of causes. Computations derived from data presented in Table 3

CATEGORY OF CAUSES OF STILLBIRTH	NUMBER	MASCULINITY RATE	SEX RATIO
Malformations	10,301	42.8	748.9
Thrombosis of cord	331	51.7	1068.8
Extra-uterine pregnancy	86	52.3	1097.6
Predisposition of mother to abortion	93	53.8	1162.8
Albuminuria and other diseases incident to pregnancy	11,721	54.1	1176.2
Hydramnios	1,043	54.7	1205.1
Other specified causes	4,516	56.4	1295.9
Antepartum hemorrhage	69	56.5	1300.0
Inertia of uterus	350	56.6	1302.6
Death in utero	7,400	56.6	1304.6
Rupture of uterus	241	56.9	1317.3
General diseases	6,695	57.0	1325.5
Diseases of the placenta and membranes	19,087	57.0	1326.8
Other diseases or conditions of mother	4,077	57.0	1327.1
Causes not specified or unknown	45,302	57.4	1346.1
Asphyxia of child (cause not stated)	7,411	57.9	1373.8
Prolapse and compression of cord	17,855	58.2	1392.1
Hemorrhage of uterus	446	58.3	1397.8
Malpresentation	9,036	58.9	1433.6
Abortion, miscarriage, premature birth	13,296	59.4	1464.5
Traumatism and overwork	5,295	59.6	1476.6
Difficult labor	16,773	62.7	1628.8
Prolapse of uterus	48	70.8	2428.6
Retroversion of uterus	76	71.1	2454.5

portion of the female deaths. The order of importance of the causes of stillbirths is somewhat different for the two sexes and while it might seem that there is not any great divergence between the males and females with respect to relative frequency of each cause, the sum total of the discrepancies between the two distributions is such that chi-square equals 1204.8. This indicates a difference statistically significant since the probability of its being due to chance alone is exceedingly small. Among the causes with a different incidence in the two sexes are to be noted Malformations with a relative frequency in the females almost twice as high as in the males and the diseases classed under Albuminuria and other diseases incident to pregnancy which occur relatively more often in the females. The discrepancies between the two distributions are increased in the other direction by the incidence of Difficult labor, Traumatism and overwork, and Malpresentation, all much higher in the males.

The above differences are reflected in the distribution of the masculinity rate and sex ratio of the stillbirths from each cause (cf. Table 4). Masculinity is lowest for Malformations, in particular for Spina bifida for which the sex ratio is 560 and "Other" malformations with a sex ratio of 624, while for Hydrocephalus, the sex ratio equals 1108. Among the important causes the highest sex ratio occurs in stillbirths due to Difficult labor — it reaches a maximum of 2259 for stillbirths accompanying Version. When the usual statistical procedure is applied to determine whether the fluctuations of masculinity rates for each cause category can be regarded as chance deviations from the masculinity rate for all causes, it is found that the masculinity rates for Malformations and Albuminuria *et al.* are significantly lower than that for the total of all causes. The first category includes Hydrocephalus, Spina bifida and "Other" malformations. For all these subdivisions the masculinity rate is significantly lower than that of all causes. The same is true for stillbirths due to Albuminuria, Toxaemia and Eclampsia, subdivisions of the Albuminuria *et al.* category, but it is not true for stillbirths due to Nephritis which are also included under this general heading. The major categories of causes of stillbirths which have a masculinity rate significantly above that of the total are Abortions *et al.* (but not the subdivision "Other premature"), Prolapse and compression of cord (true only for Prolapse), Malpresentation, Traumatism and overwork (excluding blow, fall, shock and overwork) and Difficult labor. The last class includes stillbirths associated with Deformed pelvis,

Instrumental delivery, Version and "Other"; for all of these the masculinity is significantly high compared to that of the total.

The causes of intrauterine mortality as indicated by stillbirth, on the whole, are definitely different in the two sexes. Consequently it would appear that the high masculinity of stillbirths is not so much a manifestation of an absolute inherent fragility of the male but rather of the liability of the male to certain specific pathologic factors which are prevalent as causes of stillbirth. But, before discussing further these findings, it is important to know whether they are interrelated with the changes in masculinity which accompany the length of the uterogestation period.

MASCULINITY, CAUSE OF STILLBIRTH, AND DURATION OF UTEROGESTATION

In Table 5 are presented the more important categories of causes of stillbirth in ascending order of masculinity and for each category is also given the percentage distribution of the stillbirths according to duration of uterogestation. In order to keep the numbers fairly large, this last item has been classified in the three usual divisions; 1-4 months, 5-7 months, and 8 months and over.

Since it is here intended to arrive at a measure of the variation of masculinity in terms of the cause of stillbirth, only those categories that include stillbirths for which the diagnosis was more or less specific have been selected. Table 5 presents information of considerable importance for this inquiry. In the first place it is seen that increase in the total masculinity rate does not follow regularly an increase or decrease in the percentages of the stillbirths that have occurred at 8 months and over, at 5-7 months, or, at 4 months and under. This means that the rank occupied by each category of causes is not consistently affected by whatever differences relative to duration of uterogestation there might exist among the categories of the causes of stillbirths.

Secondly, the rank occupied by each category of causes of stillbirth remains fairly stable in the three uterogestation periods. The rank correlation between the position of each category of causes relative to its masculinity rate in the 4th month and less period and in the 5th-7th month period equals $+.74 \pm .10$; between that in the latter period and in the 8th month and over period it equals $+.53 \pm .15$; and between the order in the first and in the last period it equals $+.52 \pm .15$. All of these coefficients are probably significantly different from zero. The

greatest discrepancies found in the relative position of each category of causes in successive uterogestation periods occur for the category of Abortions (which has a high masculinity rate in the period of less than 4 months and declines thereafter) and for the category of Difficult labor (which shows a comparatively low masculinity rate in the 5th and 7th

TABLE 5

Masculinity rates and distribution of stillbirths according to duration of uterogestation for the more important categories of causes of foetal mortality

CATEGORY OF CAUSES	MASCULINITY RATES				NO. OF STILLBIRTHS	PERCENT OF STILLBIRTHS IN SPECIFIED PERIOD			
	Total	8 mos. and over	5-7 mos.	4 mos. and under		8 mos. and over	5-7 mos.	4 mos. and under	Total
Malform.	42.8	44.4	38.5	49.0	9,262	73.1	26.4	0.5	100.0
Album., etc.	53.9	55.9	51.5	61.6	11,031	49.6	47.7	2.7	100.0
Death in ut.	56.7	56.5	55.7	68.2	6,969	59.8	35.5	4.7	100.0
Gen. diseases	57.0	56.4	56.8	65.8	6,255	49.8	44.1	6.1	100.0
Dis. plac. and memb.	57.1	56.5	56.6	70.0	18,144	55.3	40.4	4.2	99.9
Asphyxia	58.0	57.4	56.9	74.8	6,495	79.0	17.0	4.0	100.0
Prol., comp. cord	58.1	58.2	56.7	65.8	16,756	86.9	12.2	.9	100.0
Malpresent.	58.8	58.8	58.7	79.2	8,357	92.1	7.7	.3	100.1
Abortion, etc.	59.3	54.1	56.8	71.4	12,040	10.9	69.9	19.2	100.0
Trauma., etc.	59.6	57.8	57.4	69.8	5,116	29.6	54.2	16.2	100.0
Diffic. lab.	62.7	62.9	55.9	70.0	15,755	96.9	3.0	.1	100.0
ALL SPECIFIED CAUSES	56.9	57.0	54.6	69.3	127,334	63.8	31.2	5.0	100.0

month period and a high masculinity rate in the 8th month and over period). So it seems safe to conclude that the categories of causes of stillbirth when ordered according to the masculinity rate or sex ratio conserve substantially the same rank in each class of duration of uterogestation period.

Another fact to be noted from this table is that for each group of causes the pattern of masculinity relative to duration of uterogestation

is not always the same as that observed for all stillbirths. While for all the groups the masculinity rate in the fourth month and less period is always the highest, it is not always the lowest in the 5-7 month period. For the causes classified under General diseases, Diseases of placenta and membranes and under Abortions *et al.*, masculinity is higher for the stillbirths of the 8th month and over period than for those of the preceding period. This apparent reversal of the characteristic trend is due essentially to the method of grouping, the 5-7 month class being weighted considerably by the high masculinity rate for the 5th month. Instead, for each group of causes of stillbirths, the relative trend of the masculinity rate from the 5th to 9th month is as shown in Table 6.

TABLE 6

Index numbers of masculinity rates of stillbirths according to duration of uterogestation for the more important categories of causes of stillbirth (Masculinity rate of 5th month = 100)

CATEGORY OF CAUSES	INDEX NUMBER OF MASCULINITY RATES Month of uterogestation				
	5	6	7	8	9
Malformations	100	104	99	115	134
Albuminuria and other diseases incident to pregnancy	100	92	92	110	113
Death in utero	100	80	81	84	89
General diseases	100	98	82	84	91
Diseases of the placenta and membranes	100	98	93	99	94
Asphyxia of child (cause not stated)	100	104	90	103	96
Prolapse and compression of cord	100	64	74	77	84
Malpresentation	100	77	91	87	88
Abortion, miscarriage, premature birth	100	88	85	83	76
Traumatism and overwork	100	94	85	95	93
Difficult labor	100	86	92	121	121

In this table, the masculinity rate of the 5th month has been made equal to 100 and those of the following months are presented as index numbers based on the rate for the 5th month. From this table it is seen, just as has been noted for all stillbirths, that the 6th-7th month has the minimum masculinity rate and this increases for preceding and subsequent periods. The one exception is found in the case of Abor-

tions *et al.* which includes a varied assortment of so-called causes of stillbirth. The observations reported in Table 6 indicate that the differences in the distribution of the male and female stillbirths according to duration of uterogestation as noted in Table 2, in general exist independently of the causes of death, even, it is very important to note, for Malformations where the females predominate. In view of this trend the masculinity rates for each group of causes as shown in Table 5 should be corrected for differences in the distribution of the cases according to period of uterogestation. This can be done by making the distribution for each category equal to that observed for the total. The masculinity rates so corrected are given in Table 7.

TABLE 7

Corrected masculinity rates of stillbirths according to the more important causes of foetal mortality

CATEGORY OF CAUSES	CORRECTED MASCULINITY RATES
Malformations	42.7
Albuminuria and other diseases incident to pregnancy	54.8
Abortion, miscarriage, premature birth	55.8
Death in utero	56.8
General diseases	56.9
Diseases of the placenta and membranes	57.2
Asphyxia of child (cause not stated)	58.1
Prolapse and compression of cord	58.1
Traumatism and overwork	58.3
Malpresentation	59.1
Difficult labor	61.0

Comparison of the rank of the group of causes in this table with that shown in Table 5 indicates that the one fundamental change brought about by this correction regards the masculinity rate of Abortions *et al.* which has dropped considerably. This has occurred because a good proportion of the stillborn at 4 months or less have been eliminated and consequently the bulk of the cases listed under the general heading of Abortions are more specifically classified as "Other premature". With this exception, the order of the groups of causes of death in terms of masculinity is practically the same in the two tables.

From Table 6 it appears then that, duration of uterogestation being made uniform, the masculinity of stillbirths and abortions varies with respect to the major groups of causes of stillbirth in the following manner:

1. Masculinity is the lowest for stillbirths said to be due to Malformations. In fact more females than males are among the stillbirths included in this group of causes and consequently the masculinity for this group equal to 42.7, is far below that of the live birth which equals 51.4 for the United States in the same period.

2. Stillbirths included in the Albuminuria group have a masculinity rate equal to 54.8 which is slightly higher than that for live births but lower than the stillbirth masculinity rate from all causes in this selected area.

3. The masculinity rates of stillbirths apparently due to General diseases such as Syphilis, Diseases of placenta and membranes, Asphyxia of child (cause not stated) and to Traumatism and overwork or those included under the category of Death in utero and Abortions ("Other premature") do not differ significantly from the masculinity rate of the stillbirths from all causes. The same is probably true of stillbirths due to Prolapse and compression of cord. The masculinity for this group of causes, it will be noted, is remarkably close to that for Asphyxia (cause not stated). These two categories presumably produce foetal death in a similar manner.

4. The two groups of causes of stillbirths associated with parturition—Malpresentation and Difficult labor—have definitely the highest masculinity rates. In the latter group are included practically twice as many males as females, the masculinity rate being equal to 61.0.

SUMMARY AND DISCUSSION

Official statistics on stillbirths and abortions collected for a selected area of the United States, when analyzed with respect to variation in masculinity relative to length of uterogestation and causes of foetal death, reveal a number of significant facts which have been apparently overlooked in the formulation of current theories about the problem of masculinity.

With regard to the proportion of males and females among the stillbirths at different periods of uterogestation, the findings indicate:

1. In agreement with observations made elsewhere, masculinity decreases as the length of uterogestation increases up to and including the

7th month at which a minimum masculinity rate or sex ratio is found. Thereafter masculinity increases.

2. This characteristic trend is the result of a sex difference in the distribution of stillbirths according to duration of uterogestation. Among the females, the percentage of stillbirths occurring before the 6th and after the 8th month is lower than that observed among the males for the same periods, while the percentage occurring between the 6th and 8th month is higher. As a consequence of this, masculinity is a minimum in this interval.

The last item is of special interest. Notwithstanding that the masculinity of stillbirths due to the several causes may differ considerably, one still finds the characteristic trend of masculinity associated with length of uterogestation. In particular it is to be noted that the trend is apparent for stillbirths due to Malformations among which the females outnumber the males, as well as for the other causes among which there are more males than females. It is also apparent for stillbirths classified as Death in utero although for these the time of expulsion from the uterus is different from that of death. In the light of these observations, it would seem that the trend of masculinity in relation to duration of uterogestation is essentially the expression of a difference of rhythm in the liability of expulsion of the foetuses of each sex. This difference may well be the result of inequality in the rate of physical development between the two sexes, but, factual basis for this or any other conclusion is not available at present.

With regard to masculinity and causes of death, the results of this analysis may be summarized as follows:

1. The relative frequency of the presumed causes of death is statistically significantly different for the stillbirths of the two sexes. Malformations and the class Albuminuria and other diseases incident to pregnancy occur relatively more often in females than in males. On the other hand, in the males is found a relatively higher incidence of stillbirths supposedly due to Difficult labor, Malpresentation and Traumatism and overwork.

2. This sex difference in the incidence of the causes of stillbirth has as a result a significant variation in the masculinity of the several causes of intrauterine mortality. A very low masculinity is found among stillbirths from Malformations, a very high masculinity among those from Difficult labor.

3. The eleven major groups of causes of stillbirth when ordered according to the value of the masculinity rate in general conserve the same rank independently of the period of uterogestation.

4. After correcting the data so as to make the length of uterogestation uniform for all groups of causes, it results that:

(a) Stillbirths said to be due to Malformations are composed preponderantly of females and thus the masculinity is markedly and significantly lower than that of live births as well as of stillbirths.

(b) Cases of intrauterine mortality associated with Albuminuria and other diseases incident to pregnancy have a masculinity rate higher than that for live births but definitely inferior to that for all stillbirths.

(c) A masculinity rate equal to that for all stillbirths is observed for the intrauterine dead due to General diseases, Diseases of placenta and membranes, Traumatism and overwork, Asphyxia of child (cause not stated), or, classified as Death in utero, and Abortions (premature). The same is probably true for stillbirths classified as due to Prolapse and compression of cord.

(d) Stillbirths caused by Malpresentation and Difficult labor have a masculinity rate definitely superior to that for all stillbirths.

This order of the causes of stillbirths in terms of masculinity suggests that females predominate when the foetal mortality is essentially the result of defective embryonic development. On the other hand masculinity is high when either the placental structure is faulty or is inadequate to safeguard the foetus from toxic elements transmitted by the mother. Finally, masculinity is highest when stillbirths result from a combination of factors among which the relative size of the foetus is very important. It would appear then that the different behavior of the males and females in relation to pathological conditions arises from a dissimilarity in constitutional make-up, especially in the rate of physical development, between the two sexes. One of the most important corollaries to be drawn from these observations is that apparently there is no evidence to justify the current assumption that the males under all conditions are more liable to stillbirth. Consequently the theories which seek to explain the so-called masculine inferiority on the basis of chromosome structure, metabolism, vitamin requirements, etc., need to be modified considerably in order to be consistent with the above findings.

The data utilized here represent only a small and probably not entirely representative sample of the total reproductive wastage. Therefore more definitive conclusions are not warranted. These findings

demonstrate, however, that even a small increase in factual knowledge can reduce to a great extent some of the confidence one might have had in elaborate and involved theories.

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TUBERCULOSIS AND CIVILIZATION

PART II. INTERPRETATION OF THE ETIOLOGICAL FACTORS IN THE EPIDEMIOLOGY OF TUBERCULOSIS[1]

(*Concluded*)

BY GEORG WOLFF

I. INTRODUCTION

IF WE wish to acquire an insight into etiological factors which have determined the progress of tuberculosis and particularly the decline over the last 50 years, it will be convenient to subject several possible factors to a closer analysis in the light of our knowledge of the pathology and epidemiology of the disease. Rates of mortality will be the safest criterion to use in the analysis, because in view of the difficulty in defining satisfactorily and separating latent and open tuberculosis, morbidity is hardly amenable to statistical treatment.

We will distinguish for the analysis: (1) *the specific factor*, determined by an exciting bacillus and the resultant natural and artificial processes of immunization; (2) *hereditary factors*, determined through a suitable disposition of the host, the infected individual, as well as by the character of the population to which he belongs, indicated in some sense by language, nationality or race; (3) *the social factor*, determined by the unstable and easily varied conditions of human society. We may subdivide this latter category as between the more *socio-economic* (housing, nutrition, occupation, place of residence, whether in country or town, industrial or agrarian states, etc.) and the more *socio-biological*

[1] From the Department of Biology of the School of Hygiene and Public Health of the Johns Hopkins University. The substance of this paper formed the second of a series of three special lectures at the London School of Hygiene and Tropical Medicine, delivered in October, 1937. Part 1 of the series appeared in HUMAN BIOLOGY, Vol. 10, pp. 106-123, 1938. The numbering of the tables in this concluding part continues the numbering of the first part.

components. Of the the latter one, namely (4) *the population factor* (the fall of the birth rate, diminution in size of family, migration) is of such importance as to warrant special discussion. Finally, treated separately is (5) *the medical prophylactic factor,* which in the sense of mass prophylaxis is a function of medical knowledge and of medical treatment. Strictly speaking these (4 and 5) also come under the terms of social environment but since they differ greatly from the earlier items, their separate treatment in a study intended primarily for medical men and hygienists may be deemed reasonable.

There have been many investigations (*cf.* 10-18, for example) concerned with the decline in the rate of mortality from tuberculosis, but one does not find in them clear differentiation and separation of material according to biological and epidemiological points of view.

A still further factor is that of *climate* (that is to say an influence of natural as distinct from social environment). It is possible that some importance in treatment of individual cases does attach to this factor. But in epidemiological studies in one and the same country it can be ignored, for an alteration in climate in the short period of history to which our epidemiological studies refer is hardly thinkable. This factor may all the more readily be ignored because the influence of climate can be more than compensated for by social and economic factors. Both in the north and the south there are countries with high and low rates of mortality from tuberculosis, as is well known. Even in Switzerland, in spite of a climate favored by so many sanatoria, the mean rate of mortality of the indigenous Swiss population from tuberculosis is not particularly low. On the plains of Denmark and Holland we have a population with very favorable rates of mortality, but the French and Hungarian position is again less favorable.

We may pass now to the discussion of the single factors.

II. THE SPECIFIC FACTOR

In the etiology of tuberculosis the bacillus plays a dominating part inasmuch as without its presence bacillary infection cannot arise. The establishment of this fact we owe to the investigations of Robert Koch, but the influence of the *materies morbi* in the pathology of the true cause is not the same thing as its importance in the epidemiology of tuberculosis as a mass phenomenon. In epidemiological manifestations of tuberculosis other factors, especially social, play a part and tend to diminish the importance of *materies morbi.* Following the discovery

of the causal agent of tuberculosis, there has developed, as a further result of biological investigations, knowledge of the ubiquity of the tubercle bacilli.

In the present state of serological and pathological knowledge it would appear to be established that the specific organism has had contact with the whole population of urbanized areas. Hence it follows that the *materies morbi* by itself is not sufficient to produce clinical illness, still less an epidemic spread. In other words, in the rise and fall of tuberculosis as a mass phenomenon the rôle of the bacillus and of infection have not been so overwhelmingly important, as, for example, in the epidemic spread of acute diseases such as influenza, smallpox, measles, scarlet fever, typhus and diphtheria; here also no doubt favoring factors are present, but as compared with the specific infective factor they have been less important.

In this connection we must raise the question as to whether the fall in mortality from tuberculosis should be attributed to a general epidemic saturation[2] of civilized races, that is to a natural protective inoculation, or even to artificial protections which are analogous with smallpox vaccination as conferred on the community. On the question of the natural inoculation F. Neufeld (19), veteran Director of the Robert Koch Institute in Berlin in his general report of the tuberculosis congress at Bad Elster in 1921 reached this conclusion:

> Provisionally we must content ourselves with a natural inoculation due to an early infection with tuberculosis which, at any rate in the wider circles of the population, happens to a majority in the age of childhood. This natural inoculation produces a very limited immunity, an immunity which is only relative and rapidly diminishes. Any other immunity in connection with tuberculosis does not exist.

This cautious standpoint in the research work on immunity agrees approximately with the experience of clinicians and epidemiologists; for the typically slow course of the disease makes it clear the infection does not carry with it specific immunity. How easily the "relative" immunity of which Neufeld speaks may be broken when other factors reducing resistance are present, the rates of mortality during the war give painful proof. (See Table 4 in Part I.)

That in normal times this epidemic saturation or natural inoculation is of far less importance than other factors is indicated in a detailed study by Greenwood and Wolff (20) respecting the cities of London, Berlin and Paris. Here the epidemiological statistics confirm the results

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[2] In English there is no precise equivalent to the Germanic *Durchseuchung*—but perhaps 'epidemic saturation' may serve.

of experimental epidemiological investigations, such as those carried out by Neufeld in the field of tuberculosis, and recently by numerous fellow-workers of his school. The fact that in cities the densely populated quarters show regularly higher rates of mortality than the more sparsely populated areas is an argument against the practical importance of epidemic saturation, or at least an argument that relative immunity falls far behind the social factor in importance.

With regard to the second question as to whether artificial immunity, perhaps by vaccination, is in a position essentially to change the course of the epidemiology of tuberculosis Neufeld again remarks:

> We should not forget that in all processes of immunization we are doing nothing else but imitating nature, as, for instance, is shown by the observation of antitoxin in the blood of the convalescent who is recovering from diphtheria, or of the bacteriolytic substance in one recovered from cholera. Nature presents to a person who has passed through an attack of smallpox or measles an immunity which may be life-long but usually does not make this gift to the person who has passed through an attack of tuberculosis. Who has for a goal, as do some investigators of to-day, the hope of reaching in tuberculosis the kind of immunity that is obtained in smallpox, is hoping for something which simply does not exist in the facts of life.

We have nothing to add to this conclusion of an authority in the field of immunology. His criticism of protective inoculation is completely consistent with what has been inferred by epidemiological statisticians. But when a man of the position of Calmette (21 and 21a) who has devoted his whole life to the experimental study of tuberculosis, reaches the belief that in BCG (*Bacilli Calmette-Guérin*) a practical means of protection has been found and claims support all over the world, the subject deserves a further word. The errors of Calmette's statistical treatment need no further consideration, adequate attention having already been called to them by Greenwood (22) and Rosenfeld (23). Here I shall refer only to certain logical and epidemiological aspects. Calmette thinks that by the peroral administration of protective substances, the infant mortality of children brought up in a tubercular environment has been reduced to one-third of the rate observed elsewhere in France. That is to say, an infant mortality of 3 percent of live births vaccinated as compared with the figure in France at the time of the inoculation, 8.3 in 1927 and 9.1 in 1928. Accordingly it would appear that in BCG he had a means of combating not only tuberculosis in infants but all forms of mortality. Now let us consider the corresponding figures for Greater Berlin at the same time. In 1928

of 43,352 live births 3379 died between the age of 0 and 1; 68 of these were from tuberculosis, that is approximately 2 percent of the deaths. By far the larger number died from premature birth and general debility, birth injury, inflammation of the lungs, and defects of nutrition. These are the most important causes of death in infancy. So we have an infant mortality calculated in the usual way from the above figures of 7.8, and without tuberculosis of 7.6 per 100 live births. So that in the ideal case that in Berlin, whether by inoculation or whether by other means, not a single death from tuberculosis occurred, the influence on the general figure of mortality during the first year of life would be wholly insignificant. Indeed as the figures for Zürich in Table 5 (Part I) show, the share of tuberculosis mortality in the first year of life is there also very small; it amounted in 1926-33 to 1.7 percent for males and 2.4 percent for females of the total mortality. That is about the same as in Berlin.

One could, therefore, never hope with the most successful prophylactic against tuberculosis for such an effect as Calmette has claimed. Are we to suppose that this method also prevents mortality from general debility, premature birth, etc., a rather remarkable consequence of peroral administration of an avirulent strain of bacilli? It is noteworthy that in France neither infant mortality nor tuberculosis in general has been particularly low of recent years; we can only repeat the words of Neufeld that one is assuming a phantom if one seeks to regard immunity against tuberculosis as in the same category of ideas as immunity against smallpox. Immunity in tuberculosis, in the sense of the classical antibody theories, does not exist.

Even immunity through a relative epidemiological saturation does not afford a sufficient protection; therefore, on these lines we cannot explain completely the fall of mortality from tuberculosis (24 and 24a).

III. HEREDITARY FACTORS

A few words must be said on the relation of the hereditary factors to the epidemiology of tuberculosis. That a constitutional factor plays its part in any disease should be self-evident. That from the bodily habitus and constitution, in the Greek sense, some are more pre-disposed than others to tuberculosis is possible but unproven. It is undoubted that in the same family under approximately identical environments we see children who require and succumb to tuberculosis while others do not.

a. Study of Pedigrees and Twins

Heinrich Münter (25) has devoted special attention to this problem of the varying reaction of members in the same family to tuberculosis. He has attempted on genetic lines to reach an explanation on the hypothesis of a simple recessive inheritance: those members of the family remaining healthy are assumed to be heterozygotic, not showing the recessive character, but conveying it to the following generation. Those suffering from open tuberculosis are homozygotic and have received the factor from both the parents; these can be recognized because of the open manifestations of the disease and should be eliminated by sterilization. It is easy to see, however, from Münter's own tables which are carried back to the beginning of the nineteenth and even to the eighteenth century, that his assumptions are frequently arbitrary. He accepts the deaths from tuberculosis without any official refinement even when death occurred in middle age and there was no other diagnosis. Further he makes the case easier for himself by assuming not a specific factor but an organ predisposed for infectious diseases of the lungs as a whole including pneumonia, bronchitis, asthma, etc. With such simplifications it is certainly not difficult to find specious evidence of a recessive inheritance.

It is true enough that this investigation again called attention to the fact well-known in clinical experience, that members of the same family do react differently. This being a fact it is not surprising that the study of twins leads to similar conclusions. Following a method introduced by Galton, Diehl and v. Verschuer (26) made a careful study of the pathology of tuberculosis in monovular and binovular twins. They studied 127 pairs of twins in which one, both, or neither of the twins suffered from tuberculosis, and have shown certain differences. This is undoubtedly of value, for their results are unambiguous, even if certain reservations on the question of method have to be made. (Diagnosis of similarity in the hereditary constitution of the twins and the diagnosis of similarity in the judgment of concordance or discordance as to tuberculosis and the environment by the same investigators.) The authors excluded 21 pairs in which no distinct evidence of tuberculosis was obtainable and with the remainder have reached the conclusions shown in Table 9.

Accepting all the conclusions of the authors on the classifications, we find that there is a difference, on admittedly small numbers, of 45 percent $\pm$ 9.1. We can conclude that there is a real difference in the behavior

TABLE 9

Behavior of monovular and binovular twins in respect of tuberculosis

KIND OF TWINS	BEHAVIOR IN RESPECT OF TUBERCULOSIS	
	Concordant	*Discordant*
37 identical pairs (monovular)	26 = 70% ± 7.5	11 = 30% ± 7.5
69 non-identical pairs (binovular)	17 = 25% ± 5.2	52 = 75% ± 5.2

of identical and fraternal twins in respect of tuberculosis. Identical twins behave in regard to incidence of tuberculosis, more uniformly than merely fraternal pairs. But can we infer from this that tuberculosis is an inherited disease, or that a 'constitutional state' is of "decisive importance for the origin and course of tuberculosis"—a view reached by the authors in the sense of distinguishing a "tuberculosis gene" as a basis of predisposition? Such an identification appears to involve a logical fallacy, jumping over a whole series of members of the logical chain (Redeker 27). The authors have overlooked the fact that genetically identical individuals, by the mere fact of this identity, must in their reactions to acute infectious diseases, as well as to chronic diseases, or even to lead poisoning, which is surely an exogenous injury, behave more uniformly than genetically non-identical individuals. Nobody suggests the existence of a specific typhus gene or typhoid gene, although as any experienced observer knows those acute infections in one and the same epidemic pursue a different course in different individuals. Tuberculosis is also an infectious disease conditioned by the epidemic *materies morbi*, which is a *conditio sine qua non*. From this result of Robert Koch's it is believed that neither the student of heredity nor the student of environment can dissent.

It is certainly indisputable that inherited factors form a general constitution and play an important part in the pathology of most diseases. But a precise differentiation of what are the special characteristics of hereditary qualities predisposed to tuberculosis or what is the kind of constitution affording protection against it has not, unfortunately, been discovered by the investigation of pedigrees or through the examination of twins. On the other hand, the investigation of siblings by Diehl and v. Verschuer, using twin material, have confirmed what was the

starting-point of Münter's investigations of pedigrees, namely the different reactions of siblings or of non-identical twins in the presence of tuberculosis.

Pearl (28) in an instructive chapter on Constitution and Tuberculosis (*Studies in Human Biology*) dealt statistically with family histories of individuals afflicted with tuberculosis, and of others free from tuberculosis, both sets of individuals being taken from the same economic and social strata. Proving first the occurrence of tuberculosis among the blood relatives of 38 tuberculous and 19 non-tuberculous persons with more than 5000 blood relatives he found a much higher attack rate among the relatives of a tuberculous person than of a non-tuberculous person, as was to be expected. Taking all the generations together (same generation, parental, grandparental, great-grandparental, child generation) this family material showed that a tuberculous subject had nearly six times as many blood relatives tuberculous (7.0 percent) as had a non-tuberculous person (1.2 percent). But then he examined carefully the duration and extent of personal contact of individuals in the family history with tuberculous persons, whether in the family home or outside. Taking those offspring, whether tuberculous or not, that were known to have been in close contact with an active case or *not* to have been he got, on 690 individuals for whom the contact relations could be found, the results shown in Table 10.

TABLE 10

Percentage of close contact in different ancestral groups
(*After Pearl*)

ANCESTRY	PERCENT OF TOTAL OFFSPRING TUBERCULOUS	PERCENT OF	
		TUBERCULOUS OFFSPRING IN CLOSE CONTACT	NON-TUBERCULOUS
No parent or grandparent tuberculous	7.4	22.2	11.2
No parent, one or more grandparents tuberculous	17.3	41.2	19.8
One parent, and no, one or more grandparents tuberculous	19.8	94.1	73.9
Two parents, and no, or one grandparent tuberculous	38.2	95.2	76.5

"These results", writes Pearl, "are a striking illustration of the caution which must be exercised in drawing conclusions about the inheritance factor in the etiology of tuberculosis." The figures show very distinctly that the attack rates of tuberculosis among the offspring increase very much with the increasing amount of tuberculosis in the direct ancestry, but the rate of close contact "increases enormously more rapidly than does the rate of incidence" and is furthermore in each of the four ancestral groups higher for the tuberculous offspring than it is for the non-tuberculous offspring. On the other hand, the figures in Pearl's investigation, that should be consulted in the original, show equally that where one or both of the parents were actively tuberculous, *"virtually three-fourths of the non-tuberculous offspring have been in just as close contact with active open cases as their brothers and sisters who unfortunately developed the disease."*

These statistical results from united family histories are in good accord with our clinical experience. They considerably antedated the similar results of Heinrich Münter's work on single pedigrees and the studies of tuberculosis in monovular and binovular twins above-mentioned. But Pearl is much more cautious, demanding in the first place more accurate data in this field of research before drawing any conclusions when he writes towards the close:

> The disturbing thing, and the thing which demonstrates that much further work must be done upon the problem, is that in every case the close contact rate is higher for the tuberculous offspring than it is for the non-tuberculous offspring. In other words, these figures indicate that familial contact with active open cases is beyond question a factor in determining the incidence rate of clinically active tuberculosis. It appears equally obvious, however, that it certainly does not account for the whole of the increase in the incidence of the disease which we find to occur as the amount of tuberculosis in the immediate direct ancestry increases.

Pearl in a later paper (29) although accepting the importance of hereditary factors shows how impossible it is by the means of any simple Mendelian schema to interpret the hereditary factor in tuberculosis. Wilhelm Weinberg (30) in his actuarial study of *The Children of the Tuberculous* shows that the offspring of tuberculous parents have an increased attack-rate of tuberculosis, in conditions where father or mother or both parents were affected. Weinberg using the method of life tables for the period 1873-1902, with numerous sub-divisions in accordance with the age at death of parents, birth interval of children with the death of parents, the number of children and birth year, and the social status of parents, finds an increase in general and tuberculosis

mortality of children with a family environment of tuberculosis: Braeuning and Neumann (31), and Peller and Bettelheim (32) published similar results. Pearl working again on data from families, not arbitrarily selected, reached the following conclusions, using the material of 564 pairs of parents and 2480 children. Putting the frequency of open tuberculosis in the children of parents neither of whom has clinical disease as equal to one, then, if the father alone was tuberculous the relative incidence figure was 1.7; if the mother alone was tuberculous, 1.6; if both parents were affected, 4.3. Whether, as Weinberg thinks, it is an increased danger of infection in families associated with social factors, or, as Pearl concludes, the influence of genetic factors, particularly when both sides of the family are involved, can hardly be determined.

That the hereditary factor plays an important part may also be inferred from the fact that open tuberculosis also occurs in the well-to-do classes, if not so frequently, as among the less well-to-do.

b. Tuberculosis and Natural Selection

As a corollary to these remarks on hereditary factors something must be said on the question of whether the endogenous constitution of the whole population has any influence on the epidemiology of tuberculosis as a mass phenomenon. The epidemiological events of the last decennium speak decidedly against this, and rather tend to show that from the whole complex of causal factors, variable factors of the social environment have most influence on the march of mortality from tuberculosis. The decline of tuberculosis, which rose again in the war years, speaks not only of the importance of the social conditions, particularly national nutrition, but also of population factors and medical treatment in these years of scarcity. The other essential conditions, the virus infection, the hereditary quality of the population, the climatic conditions, can hardly have varied in this short space of time and might be regarded as constant.

Some authors have recently again brought the theory of selection by mortality into the foreground, the view which long ago Karl Pearson (33) used to explain the decline in mortality from tuberculosis. But this natural selection by the early death of the tuberculous does not sufficiently explain the epidemic course. The reproductive powers of the affected in a majority of cases continue until death and secure a maintenance of their stock.

A particularly enthusiastic supporter of the doctrine among German writers is Fritz Lenz (34) who claims, among other things, that there is a particular relation between susceptibility to tuberculosis and skin pigmentation. On the basis of the investigations of a few earlier writers, he comes to the conclusion that blonde types are less affected by tuberculosis than brunettes. "If this is the case", he writes in his well-known and frequently printed *Menschliche Auslese und Rassenhygiene*, "one must seriously believe that tuberculosis has been a contributory factor in producing in North Europe, poor in sunlight, the blonde clear-skinned type, while in the South, where the blonde type is more susceptible to malaria, there has been a change over in favor of the brunettes." Gottstein (35) in a book which appeared recently remarks that epidemiological data point in the other direction. In high altitudes, in Sweden and Norway, mortality from tuberculosis tends to increase, which may well be correlated with the long winter, lack of sunlight, and a consequent frequency of overcrowding. Lenz adds "I think it not improbable that the remarkable frequency of blonde individuals among Jews in Central Europe, which has given anthropologists so much difficulty, is due to the favoring of the blonde elements by the selective action of tuberculosis." One can of course theorize, but here more exact knowledge of the epidemiological data suggests that the lower mortality of Jews from tuberculosis is correlated with economic factors (see Goldman and Wolff, 36). We will not carry this further. As we cannot alter hereditary constitution or pigmentation of hair and skin, it will be more practicable to carry out a campaign against tuberculosis where it might be effectual, i.e. against factors of social environment which are, for the most part, the work of man. This brings us to the last part of the epidemiological analysis.

IV. THE FACTOR OF SOCIAL ENVIRONMENT

The relation of the social factor as a whole to the course and the distribution of tuberculosis is to-day in little danger of being underrated; too many special investigations have measured the influence of the environment. It has often happened that the particular components which together form the social factor have been arbitrarily assessed. If we wish to proceed with the logical and statistical analysis we shall find it convenient to separate the complex social environment into (a) the socio-economic factor, (b) the socio-biological factor, and (c) the medical and preventive factor.

a. The Socio-economic Factor (Tuberculosis and Well-being)

By this we are to understand the influence of well-being and social status in general on the course of the epidemiology of tuberculosis. They are, in fact, highly correlated. This factor of economic well-being itself can be analyzed into series or parts (housing, nutrition, occupation, situation in the occupation, size of family, hygienic education, etc.) which are highly correlated one with another. Thus when the rate of mortality from tuberculosis is found to be highly correlated with the measure of domestic overcrowding it is only in a limited sense correct to draw the conclusion that tuberculosis is a "house disease". The conclusion is only correct on the understanding that overcrowding is to be regarded as an index of general social conditions.

Of such indices of well-being there are naturally many, including such direct ones as income, for instance, but these are only rarely obtainable from or in connection with statistical data regarding deaths. For that reason indirect indices have generally been used: Hersch (37 and 37a) uses as an index of the poverty factor of the population the proportion in an administrative district not assessed to a house tax and finds a striking regularity of relation between tuberculosis mortality and the index of poverty in the different districts of Paris; thus in the period 1911-13 mortality from tuberculosis tended to vary as the square of the index of poverty, leading Hersch to the conclusion that mortality from tuberculosis was "an inseparable attribute to poverty" (admitting it to be a disease which often enough appears among the well-to-do but among them more often tends to recovery). Although so simple a mathematical relation between mortality of tuberculosis and social position could never have general validity, as the definition of 'poverty' in the different countries and cities must vary with the indices chosen, still it is true, that the correlation is very high, especially in Paris. The mortality rate from tuberculosis in the *arrondissements* rises and falls more steeply than the corresponding indices of poverty. Calculating the coefficient of correlation Hersch reaches the figure of $.97 \pm .02$. Subsequent tests for post-war experience by Greenwood and Wolff for the year 1921-22, and by Hersch himself for 1924-28, showed that in Paris this relation subsisted.

Other investigators have used other indices of economic well-being. Stevenson (38) took the number of domestic servants in London; Greenwood and Wolff (20) for Berlin the proportion of manual laborers and of domestics; Wolff and Jahn (39) the proportion of manual

laborers; Prinzing (40) for Vienna, and Wolff (41) for London, the index of overcrowding. (Table 11.) Winkler (42) found for Charlottenburg, which is now a part of Greater Berlin, that the number of deaths from pulmonary tuberculosis was highly correlated with income. This also appeared from the data of the great insurance companies in Germany such as the Gotha Company. This direct relation between mortality and social or economic well-being has been dealt with particularly in the English official statistics on occupation.

TABLE 11

Correlation between tuberculosis mortality and economic status

PLACE AND TIME		INDEX OF WELL-BEING	COEFFICIENT OF CORRELATION	INVESTIGATOR
Paris	1911/13	Proportion of population	0.97 ±0.02	Hersch
	1921/22	not assessed with a house	0.93 ±0.04	Greenwood and Wolff
	1924/28	tax	0.86 ±0.06	Hersch
Berlin	1924/25	Proportion of workers	0.736±0.103	Greenwood and Wolff
	1924/25	Proportion of domestic servants	—0.826±0.071	Greenwood and Wolff
	1926/27	Proportion of workers	0.791±0.088	Wolff and Jahn
	1928/29	Proportion of workers	0.819±0.078	Wolff and Jahn
London	1908/12	Overcrowding (more than	0.832±0.058	Wolff
	1921/23	two persons per room)	0.774±0.075	Wolff
Vienna	1897	Overcrowding (more than three persons per room)	0.892±0.047	Prinzing

All these investigations (and many others) bring out invariably the very high inverse correlation between mortality from tuberculosis and well-being, whatever index for the latter is chosen. There is, therefore, no doubt of the close relationship between the event of an attack of tuberculosis and economic status. This is well illustrated by the unsurpassed occupational statistics of England. The most recent available relate to the years 1921-23. For the previous decennium and for 1921-23 Stevenson (43) distributed occupied males into five socio-economic classes, taking into account the differences in age composition by means of the simple and ingenious method of choosing the population

at ages, such that at the rates of mortality within these age groups observed, for all occupied and retired males, the deaths would number 1000. When the actual rates of mortality in a particular group are compared with this frame-work the resultant number of deaths can be directly compared with 1000. Table 12 illustrates the results where Class I means the highest social class, and Class V the lowest—that of unskilled laborers (43, 44).

TABLE 12

Mortality from tuberculosis, cancer, diabetes and all causes of death in the English male population by social classes, 1921-23
(Standard figures)

CAUSE OF DEATH	CLASS I	CLASS II	CLASS III	CLASS IV	CLASS V	ALL OCCUPATIONS AND CLASSES TOGETHER
Tuberculosis	90.1	151.6	173.4	177.7	243.8	177.3
Cancer	102.5	118.1	127.1	123.8	157.8	128.4
Diabetes	15.2	17.7	11.2	9.2	8.1	12.2
ALL DEATHS	812	942	951	1007	1258	1000

These few figures are very instructive. We recognize some increase from class to class in general mortality, and a more irregular change in cancer mortality, but the change in respect to tuberculosis leaps to the eye. Already in Class III (skilled laborers) and in Class IV (intermediate) the rate is twice, and in Class V (unskilled laborers) three times that in the small highest social and occupational stratum. Even in Class II (intermediate) it is already appreciably higher than in Class I. Mortality from diabetes, on the other hand, follows the opposite rule. Here the standardized figures of Classes I and II are almost double those in the lowest social classes. These observations running through the social classes of the English occupational statistics throw some doubt upon the interpretation of the excess mortality from diabetes in Jews and on their subnormal mortality from tuberculosis. It is evident before attributing importance to racial factors that attention must be given to socio-economic factors (Goldman and Wolff).

b. *Housing and Tuberculosis*

I shall refer only briefly to the individual components of the complex of social environment. In general, statistical data do not allow the isolation of the particular factor of housing, and as already noted, the high correlation between overcrowding and tuberculosis does not alone suffice to incriminate housing above all other social factors.

If we wish to measure the special influence of dwellings in favoring infection we must endeavor to eliminate all other factors, the statistical attempt to do this being by the so-called partial correlation coefficient technique. A complete elimination of all other social factors which go to make up the attributes of poverty that favor tuberculosis, is exceedingly difficult; one might perhaps consider the housing conditions of groups of the population at the same level of income, but even then there are inequalities which are of importance, as for instance, occupation, size of family, hygienic education. A bad or insufficient house is always only a part of poverty. In general one might venture to say that the influence of housing on the spread of tuberculosis has been exaggerated. In favor of this view is the fact that, in spite of the enormous housing scarcity in the post-war period in Germany, the rate of mortality from tuberculosis continued to fall. At the very least there must have been other factors which compensated the unfavorable influence of domestic overcrowding.

c. *Nutrition and Tuberculosis*

By a similar process of logical deduction, which must always be fundamental in statistical investigations, we reach the conclusion that national nutrition is of much greater importance in the epidemiology of tuberculosis. Mass experiments of nutrition in war times speak with no uncertain voice. Of the numerous components of the social factor none has so much importance as the great deterioration in the average diet of the people. In comparison with this, housing, occupation, and size of family can be regarded as relatively constant. Malnutrition, to which perhaps might be added medical treatment of inferior quality and some psychological components, played a great part in the rapid increase of mortality from tuberculosis. Even as early as 1915, in England as well as in Germany, mortality was higher than in 1913 and 1914. In Prussia the peak reached first actually in 1918 (see Table 4 in Part I) was maintained at almost the same height in 1919. That the factor of nutrition is here of primary importance at once suggests itself.

After all statistical observation of a people is in the last resort a mass experiment although, of course, one not carried out under control conditions. Its scientific interpretation must be the same as for an experiment, that is to say an attempt must be made to eliminate disturbing factors. If, however, we may attribute to nutrition the chief rôle in the increase of tuberculosis mortality during the war and the decline after it, we may certainly also reasonably infer that improvement in nutrition over the last 50 years has had its part in the decline.

d. Occupation and Tuberculosis

Occupation, and perhaps even more the social correlates of occupation, play their part. On this matter English statistics of occupational mortality give exhaustive information. I have no space for detailed discussion here. Many investigations might be cited. Among German-speaking writers the work of that indefatigable student of industrial hygiene, Teleky deserves attention; among English writers the studies of Collis, and the careful investigations of Mavrogordato and Watkins-Pitchford in South Africa, which show the high risk of tuberculosis in certain industrial occupations, particularly quartz, etc., which leads to the dangerous phenomenon of silicosis associated generally with a high mortality from tuberculosis. Particularly quarry-workers and metal workers, using for sharpening tools particular mineral products suffer severely, while on the other hand chalk dust and coal dust play no part in the etiology of tuberculosis.

Taking the standardized mortality figures of the last occupational study, 1921-23 one has a tuberculosis mortality for all occupations together 164, agricultural laborers 96, bricklayers 127, miners 123, cotton-weavers 147, cotton-spinners 175, woollen-weavers 190, woollen-spinners 147, shoe factory operatives 298, tobacco factory operatives 327, hair-dressers 237, brewers 115, drivers and transport workers 215, innkeepers 200. It is not very difficult to deduce from these differences some different occupational hazards. Other factors of the social environment, budgeting of family wages and nutrition, are of great influence, but here particularly one may speak of occupational selection, which, although voluntarily undertaken, is of decisive importance. One can understand that hair-dressers, tailors, and shoemakers who are not exposed to any particular hazard still suffer from high mortality from pulmonary tuberculosis. Persons may enter these occupations who are physically unfit for others. On the other hand we see why bricklayers have a relatively

favorable mortality, as do also coal miners and brewers. These are not occupations within the power of men of poor physique. We may content ourselves with these figures but should, perhaps, add the special occupational risk of silicosis which is naturally attended by very high mortality figures from pulmonary tuberculosis. The figure for tin and copper miners is 1447; metal grinders 696, potters 450, file-cutters 399, cutters 368, gilt and metal polishers 347, stone-cutters 332, all showing figures far above the average of 164. It is also true of waiters and barmen with 440, pedlers 374, dock laborers 311, persons subjected to other factors very different from those of silicosis.

For these various reasons the summarizing in the English statistics by reducing the occupational mortality to five great social classes is of particular value. In Table 13 mortality figures at ages from tuberculosis per 100,000 living, are shown for all five social groups. Since agricultural occupations mostly show a lower figure than other industrial occupations in the same social stratum we follow Prinzing (44) in taking out of Class II the farmers and out of Class IV agricultural laborers and give them separately. This immediately gives an important comparison between agricultural and industrial rates.

TABLE 13

Tuberculosis mortality in the English male occupied population, by age and social classes per 100,000 living, 1921-23

SOCIAL CLASSES	AGE IN YEARS							
	16—	20—	25—	35—	45—	55—	65—	70+
I. Upper class	31	64	69	93	94	89	87	67
II. Intermediate class (excluding farmers)	85	149	153	159	146	122	106	67
III. Skilled laborers	89	158	142	161	171	167	132	74
IV. Intermediate class (excluding agricultural laborers)	85	163	149	194	201	184	124	86
V. Unskilled laborers	112	178	174	250	267	243	179	145
Farmers	16	58	81	76	76	77	83	39
Agricultural laborers	53	122	124	87	92	83	70	50

Mortality from tuberculosis rises almost regularly as the social class falls at all ages; there is a slight discontinuity only in Class II, the figure for which seems somewhat too high because the farmer has been taken out of the class. Farmers show a much more favorable mortality than Class II and even, with the exception of the age group 25-35, better figures than Class I. Similarly the agricultural laborers—in the same community—have much more favorable mortality than Class IV to which they economically belong.

However, in this connection we come now to a new point in tuberculosis epidemiology and economic environment, if we compare different countries as to industrialism.

e. Tuberculosis and Industrialization

We see from Table 13 the striking differences in the height of mortality in different social classes. These social differences also appear in the agricultural group inasmuch as agricularal laborers show much worse rates than farmers. This brings us to another demonstrable fact, showing a new aspect of the influence of social environment on the epidemiology of tuberculosis. In general and in any one state agricultural mortality tends to be lower than industrial, but here the influence of the general level of culture in different states makes itself felt.

For it is clear from statistical data that in spite of the risks of industrial work the average tuberculosis mortality in highly industrialized states is lower than in the agrarian states, where the standard of living and hygiene leave much to be desired. The general socio-economic and hygienic improvement of the masses in the industrial states together with their better medical treatment must be the basis of this difference. Thus, mortality from tuberculosis in the industrialized countries of England, Belgium and Germany has fallen further and is lower than in Italy and France or even in the predominantly agrarian states such as Hungary and Poland. Likewise the average mortality figure in the United States is much lower than in Japan that still must be reckoned, according to the occupational census of 1930, as primarily agrarian. Naturally there are exceptions. For instance, Denmark and Canada are states with a relatively high proportion of population engaged in agriculture but with a very high level of hygienic culture and very favorable mortality from tuberculosis (45, 46 and 47). An arithmetical summary is given by the high coefficient of correlation between industrialization and tuberculosis mortality (Table 14).

The growth of industrialization of civilized nations had brought with it not only dust and industrial diseases but also elevation of the national well-being which much over-compensates the disadvantages of industrial workers.

TABLE 14

Correlation between tuberculosis mortality and industrialization

TIME AND SIZE OF INVESTIGATION	INDEX OF INDUSTRIALIZATION	COEFFICIENT OF CORRELATION	INVESTIGATOR
1906-10 14 states of Europe	Proportion of persons engaged in industry, mining, trade and transportation	-0.647 ± 0.105	Greenwood & Wolff
1921-25 10 states of Europe		-0.766 ± 0.095	Wolff
1926-30 27 states of the world		-0.617 ± 0.119	Wolff

V. THE POPULATION FACTOR. (THE SOCIO-BIOLOGICAL COMPONENTS OF SOCIAL ENVIRONMENT)

a. The Decline in the Birth-rate and Tuberculosis

In close connection with the socio-economic development due to the industrializing of the world we have a socio-biological transformation in most industrialized states reflected in the decline of the birth-rate which set in at the turn of the century. Whether we regret this from the point of view of the quantity of the population or see in it a necessary defense reaction against threatening over-population, the fact remains that it must be considered in a study of the epidemiology of tuberculosis.

It is certain that with diminution in size of family the mean income per head has increased whether real wages have or have not risen. This self-help of individual family heads cannot have been without effect upon the economic side of the tuberculosis problem; by now the decline in the birth rate which began with relatively well-to-do classes, officials and independents, has reached the less well-to-do occupations. (Grotjahn, 48 and 48a, Burgdörfer, 49). This has been recently demonstrated particularly for England, in an impressive manner in the detailed study by W. J. Martin (50) who has followed the changes in individual counties between 1850-2 and 1930-2, making distinction between industrial and agricultural areas. The decline of the birth rate is practi-

cally universal. In the past 60 years since the period of 1970-2 this decline has been from 50-60 percent. The differences in different classes of areas have not been very characteristic. By far the greatest part of the decline has been in the last 30 years, that is since the turn of the century. This tendency was aggravated in all countries by the war and since then has hardly changed. The investigator mentioned (and many others) treats the problem primarily from the national standpoint of a particular country and, in particular, looks at the decline of births from the angle of threatening decrease of population; "social suicide" replaces the nightmare of over-population which alarmed Malthus and his contemporaries.

Recently Pearl (51) in numerous investigations has looked at the problem from the world point of view. In his last paper with Gould (52) Pearl estimates the population of the world at the beginning of 1930 to have been 2050.4 millions, and 2073.3 millions at the middle of 1931. In approximately 1.4 years there has been an increase, even in our time of some 23 millions of human beings which is a rate of increase at not quite 1 percent per annum; in the period between 1920 and 1930 it worked out at rather more than 1 percent. Estimating on this basis, a simple calculation shows that in about 70 years the population would be double (as in fact according to the calculations actually happened because the estimate of the world population in 1850 was about 1000 millions).

Such a rate of increase could not long be sustained or desired, as Pearl has (53 and 53a) demonstrated elsewhere. For that reason the decline in the birth rate of the Western civilized states should not be looked at from sentimental standpoints but rather as a mass psychological and natural phenomenon. And so we return to the problem of tuberculosis. If the influence of the social environment upon the epidemic course of tuberculosis is of importance so must transformation of the structure of the family necessarily be affected in the course of generations by the change in the birth rate, because the reduction in the size of family increases the proportion of the family budget available to each individual member; there is even a direct influence on the possibility of infection. The virus in a smaller family has a smaller field of operation, a point to which Weinberg has already called attention.

Actually statistics of most countries show a higher birth rate in general with the rise of mortality from tuberculosis, and conversely. A particularly striking modern illustration is that of Japan which, with her high live birth rate of over 30 (31.6 in 1935) per 1000 population,

TABLE 15. *Decline in birth-rate and tuberculosis mortality in European countries*

TABLE 15. *Decline in birth-rate and tuberculosis mortality in European countries*

COUNTRIES IN ORDER OF HEIGHT OF BIRTH-RATE IN 1906-10	BIRTH-RATE (LIVE) PER 1000 INHABITANTS			DECLINE, 1926-30 IN PERCENT OF 1906-10	DEATH RATE FROM TUBERCULOSIS PER 10,000 INHABITANTS			DECLINE, 1926-30 IN PERCENT OF 1906-10
	1906-10	1926-30	1933		1906-10	1926-30	1933	
1	2	3	4	5	6	7	8	9
1. Hungary	36.7	26.0	22.0	71	37.4	22.5	17.0	60
2. Austria	33.7	17.5	14.3	52	30.6	15.8	13.0	52
3. Spain	33.6	28.5	27.7	85	17.6	14.0	11.7	80
4. Italy	32.7	26.8	23.7	82	16.7	12.7	9.9	76
5. Germany	31.6	18.4	14.7	58	17.5	8.9	7.3	51
6. Finland	30.9	21.2	17.4	69	27.2	24.1	20.0	89
7. Holland	29.6	23.2	20.8	78	16.6	8.7	6.0	52
8. Denmark	28.2	19.4	17.3	69	16.2	7.6	5.8	47
9. Scotland	27.6	20.0	17.6	72	20.2	9.5	8.2	47
10. Norway	26.4	18.0	14.8	68	24.0	15.5	12.1	65
11. England and Wales	26.3	16.7	14.4	63	15.7	9.4	8.2	60
12. Switzerland	26.0	17.5	16.4	67	24.7	13.9	10.6	56
13. Sweden	25.4	15.9	13.7	63	21.1	13.1	11.5	62
14. Belgium	24.7	18.6	16.5	75	12.9	9.5	7.9	74
15. France	19.9	18.2	16.2	91	22.1	16.6	14.0	75

Coefficient of correlation between birth-rate and tuberculosis mortality 1906-10 (Columns 2 and 6) ..... $= +0.4126 \pm 0.2143$

Coefficient of correlation between birth-rate and tuberculosis mortality 1926-30 (Columns 3 and 7) ..... $= +0.2422 \pm 0.2431$

Decline of birth-rate and tuberculosis mortality (correlation coefficient) from 1906-10 to 1926-30 (Columns 5 and 9) ..... $= +0.5198 \pm 0.1884$

has a tuberculosis mortality of nearly 200 per 100,000 (193 in 1934). That there should be such a connection is in no way surprising because in general higher birth rates are observed in agrarian than in industrial states.

Table 15 gives a general view of the European countries for which comparable figures are available. If the connection between natality and mortality from tuberculosis is not striking, there can be little doubt of the intermediate link connecting economic well-being and size of family. The decline of the birth rate is as international a phenomenon as that of mortality from tuberculosis. It is not so much a question of the connection between natality and mortality that is of importance: it is rather the dynamic factor of the decline in the birth rate. If the correlations for 1906-10 and 1926-30 between natality and mortality from tuberculosis of 0.41 and 0.24 are not striking, the relation between the fall in the birth rate and mortality from tuberculosis from 15 states, given as 0.52, is of some interest. There are, however, exceptions. For instance, France, a state which has long had a low birth rate,—it is now far from being any longer the land with lowest natality—has a relatively high mortality from tuberculosis. On the other hand Holland, Denmark, and Scotland had far from the greatest declines in the birth rate, but had much greater than average declines in mortality from tuberculosis. I do not propose to enter here upon the details of a very complex problem. Statistics show merely correlations, not direct causation, but it does seem important to select from the components those which influence the epidemic course of tuberculosis in connection with industrialism, the size of family, the reduction of which is a necessary consequence of the declining birth rate.

b. Migration and Tuberculosis

Beyond the natural movement of the population by births and deaths, migration may under some conditions be of importance in the epidemiology of tuberculosis. Bradford Hill (8) in a careful study of the mortality of young adults from phthisis, has brought forward many reasons making it probable that internal migration has been of considerable importance in epidemiological phenomena. Migration from country to town, which down to the end of the nineteenth century probably brought into the towns the fittest adults, had slackened at the turn of the century. The loss of population in many rural districts had even been transformed into a gain of population. Accordingly Hill was

of the opinion that there may have been even a negative selection in the urban population, so that their tuberculosis mortality affected a population different from that of the previous century, perhaps less resistant. In agreement with this hypothesis is the fact that the excess mortality of young adults from pulmonary tuberculosis in rural districts has now disappeared. In order to get some quantitative measure—that is measuring gain or loss of population—28 county boroughs between 1921 and 1931 were correlated with the change in mortality from phthisis in young adults. In the result it appeared that there was a relation between migration rate and an increase or decrease of the mortality rate at ages 15-24. Calculated correlation coefficients of —.45 for males and —.71 for females were obtained. In rural districts a similar but less distinct relation was found.

Hill concluded, I think correctly, that in the years of his observations the level of mortality from pulmonary tuberculosis among young adults in the different rural districts was directly or indirectly influenced by the movement of the population, particularly internal migration. The urban districts, which in the last decade have attracted young people, showed on the average a decrease of mortality. Those that have lost population had on the other hand a tendency to increased mortality from pulmonary tuberculosis in young adults. One must not exclude the possibility that hereto the natural movement of the population has been of importance; notably the rise of births immediately after the war and the increase of mortality from phthisis, particularly in the female sex. That pregnancy produces a special factor of danger—well-known to the clinician—is shown in a statistical investigation by Glogauer (54) for Prussia, and by Rosenfeld (55) for Austria. They show that especially does the mortality of the female sex fall with the birth rate, but also rises with it.

In the fall in the birth rate, and changes of migration, although they are not the only factors, are probably tendencies beyond the reach of conscious will or action. The influence of migration on the change of mortality from tuberculosis in the age class particularly subject to it, is instructive. It has not been able to change the general march of mortality from tuberculosis but gives us a good example of the influence of population dynamics and the socio-biological factor.

VI. MEDICAL TREATMENT FACTOR (MEDICAL COMPONENTS OF SOCIAL ENVIRONMENT)

a. Therapeutics and Tuberculosis

It will not be denied that the successes of medical therapeutics and medical cure form a part of the social environment, but we must restrict our observations. The general figures of the epidemiologist and statistician do not and cannot replace medical initiative and practical intuition. We all admire the successes of the art of medicine which in the last decades and in all provinces have been great. We have the right to rank them high in the field of tuberculosis even if it were only the dietetic-climatic measures which in the form of sanatoria have long had their expression. But beyond this we have seen introduced internal surgical improvements, for example, in the application of pneumothorax therapy by *Forlanini,* affording a period of rest to the damaged lung—in accordance with fundamental principles of medicine—a method which has had success in seemingly desperate cases. From these and many other results the conviction has been impressed upon medical and lay minds that tuberculosis is a curable disease while its rational prophylaxis is a matter for the hygienist.

It is naturally very difficult if not impossible to appraise the results of medical methods in statistical terms. It is impossible to separate hospital isolation and other factors. As long as 30 years ago Newsholme's (10) attempt to attribute maximum importance to the selection of open cases of phthisis led to much discussion. Judgment in an individual case of treatment must be reserved for the clinician. The general conclusions as to results of treatment based upon statistics is better carried out by the clinician in cooperation with a professional statistician. Judgment respecting a particular method of treatment is more difficult in tuberculosis than in acute disease, for fatality can only be determined in hospital material with difficulty, since mild cases are insufficiently represented and compulsory notification of all cases of tuberculosis encounters many difficulties.

Compulsion to notify all forms of disease where the tubercle bacillus is present in discharges has long existed in Norway. Oslo, has therefore, individual morbidity, mortality and fatality data upon which E. Koesle (56 and 56a) has reported. The original data were published by the Oslo Health Department (57). The affected persons, not cases, were entered in a register and their subsequent history followed out

exactly. It was possible to enter the yearly acceptances of cases and the yearly decrement due to removal, cures, or death. Roesle worked out the corresponding figures for the years 1920 and 1927, which have been subsequently carried to 1931. At the beginning of 1920 the number affected was in Oslo 3564 which was reduced to 2814 in 1927, the morbidity working out to 160.4 and 135.5 per 10,000 living. The

TABLE 16

Morbidity, mortality, and fatality from tuberculosis in the city of Oslo, 1920 and 1927 (After Roesle)

	MOVEMENT OF TUBERCULOSIS PATIENTS					FATE OF TUBERCULOSIS PATIENTS		
YEAR	Stock of patients at the beginning of the year	Accession of new patients	Loss of patients by (a) moving	(b) death, i.e. mortality	Stock of patients from which death can come (2+3—4), i.e. morbidity	Proportion of cured (freed of symptoms) i.e. effect of cure	Proportion died from tuberculosis, i.e. fatality	Proportion of patients staying under observation at the end of the year
	per 1000 of the population					*per 100 of the yearly stock of patients (column 6)*		
1	2	3	4	5	6	7	8	9
				A. Both sexes				
1920	13.66	2.81	0.44	2.04	16.03	2.98	12.7	84.3
1927	11.21	2.77	0.43	1.53	13.55	6.73	11.3	82.0
				B. Males				
1920	14.65	3.13	0.55	2.13	17.23	2.30	12.3	85.4
1927	12.30	3.02	0.43	1.83	14.88	6.12	12.3	81.6
				C. Females				
1920	12.83	2.55	0.34	1.96	15.03	3.53	13.1	83.4
1927	10.31	2.56	0.42	1.27	12.45	7.33	10.2	82.5

mortality was 20.4 and 15.3 per 10,000 living, but the fatality was 12.7 percent which fell to 11.3 percent. (See Table 16).

Accordingly, the decline in mortality in Oslo is approximately equal to the decline in the number of cases. In other words there has appeared to be a real decline in the dissemination of tuberculosis which cannot be inferred from the mortality figures alone, the assumption being that

the fatality rate is relatively constant. The object of a rational campaign against tuberculosis is to reduce the attack rate; statistics of Oslo show that this has been successful in the reduction of the number of cases together with a definite increase in the proportion of cures, and consequently exits from the register. Of those with regularly equal accession rates for the two years compared of 2.8 per 1,000 of the population, the number of cures increased from 3 percent to 6.7 percent, although the fatality, particularly in males, showed little change.

Naturally these statistics which Roesle rightly describes as "the completest statistics of tuberculosis in the world" have nothing to say as to which particular method of treatment or prophylaxis is primarily represented. The modest increase of cures from 3 to 6.7 percent of all patients should perhaps be attributed to medical measures in the widest sense. As the number of new accessions has changed little in the two years it may be supposed that the important factors in the epidemiology of tuberculosis remained constant in the short period of time. Only the birth-rate has decreased in Oslo rather sharply, from 21.1 to 13.1 per 1000 inhabitants, as a remarkable fact during the period.

b. Medical Cure and Tuberculosis

On the medical cure of tuberculosis a few concluding words may be said. In all civilized states during the last 50 years the number of medical practitioners has considerably increased; in Germany from 3.9 per 10,000 inhabitants in 1876 to 7.75 in 1930 (Prinzing); in the smaller state of Austria and also in Great Britain and the United States the proportion of medical practitioners is even greater. We may infer from this that medical treatment of the population has improved in town and country. Especially is this so because social insurance such as the Sickness, Accident and Invalid Insurance of Germany which had its origin under Bismarck is now included in the widest circles of the working classes, and forms an important item of the campaign against tuberculosis. It is a matter of indifference whether this has been compulsory state insurance or the activities of private groups as in some countries. In any case it results that medical treatment is no longer a privilege of the well-to-do classes.

In connection with social insurance we have a particularly instructive study by Geissler (58 and 58a) for Baden, dealing with mortality from tuberculosis of occupied men and their dependents. They have been classified in three groups: (a) independent proprietors and managers,

(b) employees and minor public officials, and (c) working classes. The corresponding census figures for the living have been obtained from the returns for 1925. It is seen that the tuberculosis mortality rates of the different classes which used to be widely separated are levelling down. Similar conclusions have been reached by Geissler for Hessen-Nassau. I give the figures for Baden in Table 17.

TABLE 17

Mortality from tuberculosis of occupied males and their dependents in Baden, by social position of the gainfully employed, per 10,000 living in 1925

YEAR	INDEPENDENT AND MANAGERS	EMPLOYEES AND MINOR PUBLIC OFFICIALS	LABORERS
1924	9.6	9.9	15.6
1925	11.9	8.7	10.9
1926	10.4	7.0	10.2
1927	9.6	6.9	9.5
1930	8.5	7.0	7.7

There is actually here in 1930 a lower rate of mortality both of the working classes and employees as compared with the independent proprietors, which also appeared between 1925 and 1927; whereas in 1924 the excess mortality of the working classes is striking. Naturally we must guard ourselves from attributing this to "changes of the social components of tuberculosis mortality" which can hardly have varied materially in five years.

One thing at least emerges clearly from the statistics; in the insured working classes and even more among the insured black-coated workers, there is a lower risk of dying from tuberculosis, in Baden at any rate, than among those of independent position. Naturally we must ask ourselves what 'independent' means. Actually in Baden, it means farmers and peasants and persons carrying on small businesses; that is to say, classes that could not be described as well-to-do and are at certain particular disadvantages in comparison with socially insured groups when they are faced with a long illness requiring treatment. Of course there are also a few others, such as landlords and directors, in this class but

they are far too little homogeneous in respect of economic well-being to be readily compared with the other working classes and black-coated workers. The old master of German medical statistics, Prinzing (59) has remarked on this: "The middle-class has, since the war, had to face the greatest need; when illness comes he has been unable to spare himself or to make use of the sanatorium care or treatment both of which are at the disposal of the manual laborer, who comes under social insurance". We should add, perhaps, that it is precisely the black-coated worker and civil servant of intermediate grades who seek to protect themselves by keeping their families small, as is demonstrated by the most recent family statistics of Germany (60). Furthermore, since the war and inflation the working classes have followed suit, with the resulting cataclysmic fall in the birth now so much discussed. It is not that the social components of tuberculosis have changed. Indeed Geissler's research once more illustrates the extreme sensitiveness of tuberculosis to contemporaneous social factors, that is to say, to changes in the social environment. Such a change favorable to the working classes has set in. We find then that the lower rate of tuberculosis mortality among the working classes as compared with some independent classes may easily occur. But that there should be a lower tuberculosis mortality among 'poor' as opposed to 'rich' is a supposition rendered improbable by all we know of the social pathology of tuberculosis.[3]

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[3] With this point we conclude the analysis of the etiological factors in the epidemiology of tuberculosis. But logically we should perhaps add a word on the influence of *natural* environment (weather, climate, soil conditions, etc.) in contrast to the influences of *social* environment. As the natural conditions can hardly have changed in the 50 or 60 years during which tuberculosis mortality has enormously declined we need not enter into details. Climate and tuberculosis is a subject that the sanatarians and meteorological experts have discussed with zeal. One must not overlook the fact that certain climatic qualities (hours of sunshine, ultra-violet rays, etc. and other atmospheric components) are of influence on the condition of individual patients, although opinions on the subject, as for example with regard to the site and elevation of the health resort, vary greatly.

I would refer to the masterly description of Thomas Mann in his famous novel *Der Zauberberg (The Magic Mountain)* in which the influence of mountains is so much overshadowed by the special sanatorium atmosphere. Some may reject this picture, this psychological description based upon an astonishing amount of knowledge, as being only the work of a layman. But one thing at least is accepted by any physician or therapeutic optimist; the fate of a sick person is not attributed to a distant sanatorium but to the ***natural atmosphere of his home.*** This applies to the masses of sick people.

VII. SUMMARY AND CONCLUSIONS

Tuberculosis is an infectious disease; its etiology therefore requires inevitably the factor of infection. The further course of the disease is determined in each case by the congenital qualities of the individual, hereditary constitution, and various influences of his social environment. The quantitative relation of these three most important components, one to another, can hardly be determined in any individual case. Tuberculosis is, however, not an infectious disease in the sense that zymotics are infectious diseases in which *materies morbi* changes the whole foreground of the etiological picture. Married couples may live for years in close contact, brothers and sisters who have a more definite constitutional relationship with each other may live together for years, yet tuberculosis of one member of the group may not be transferred to the other members of the group. On the other hand, identical twins behave more uniformly with regard to tuberculosis infection than binovular twins, as one would expect. It is a commonplace that in other infectious diseases or external injuries, such as syphilis or lead poisoning, individuals of the same hereditary make-up must, on the average, react more uniformly than those of divergent genetic make-up. Therefore, it is a fallacy to see in this behavior of tuberculosis the character of a disease inherited by special genes. What, however, may be the hereditary factor of different dispositions, the one of which leads to tuberculosis and the other does not, has not been determined in the studies of twins or pedigrees. This is a task for the future.

In tuberculosis as a mass phenomenon of human populations, that is in the epidemiology of tuberculosis, the three above-mentioned factors play their part—the infection factor, the hereditary factor, and the social environment. The great decline in the tuberculosis mortality in the last half-century from more than 30 per 10,000 of the population to 5 or 6 affords a good opportunity to submit the factors favoring the rise and fall of tuberculosis to a critical analysis. Etiological factors in the epidemiology of tuberculosis as a mass phenomenon must be evaluated differently from etiological factors in any individual case. As over this short period of years the virus can hardly have changed sensibly, a natural immunity in tuberculosis as in some other diseases would not occur or would be easily broken (as in the hunger years of the war); one must conclude that a protective inoculation will not have any success. Again the hereditary qualities of the nations consequently must be regarded as constant over a period of 50 years, at least the positive selec-

tion by early death in tuberculosis has been made even less likely than any other alleged negative selections (due to improvement of hygiene and medicine) made by other enthusiasts. So remain as real factors above all the manifold variations and changing influences of social environment (nutrition, housing, occupation, habits of life, size of family, decline of the birth rate, migration, hygiene, therapeutics and many other influences) determining the march of tuberculosis as a mass phenomenon of society. They are inextricably combined, working sometimes together, sometimes in opposite directions and their resultant is the social factor.

Close connection between economic well-being and tuberculosis has long been known, and this social component remains unaltered. The figure of mortality from tuberculosis has fallen greatly, precisely because particular strata of the working classes are now by means of social insurance and medical treatment as well off or even better off than many of the middle classes. Here we see the supersensitiveness of tuberculosis to social influences from a new angle; the impoverished middle classes show actually less favorable figures than the socially-insured classes and black-coated workers. This tendency has been strengthened in many lands since the war by the decline in the birth rate. Any short critical examination of the statistics shows that of all the factors having influence upon the epidemic march of tuberculosis it is the socio-economic and socio-biological factor together combined with industrialization changes in the standard of living which are decisive. Some of these factors, as the involuntary experiment of the war time showed, are particularly effective; the worsening of national nutrition in the hunger years of the World War with the consequent enormous increase of mortality from tuberculosis; the maintenance of small families in the post-war period, working in opposite directions; changes of internal migration during the last decade for instance in England with, as a consequence, decided increase of mortality from tuberculosis in young adults, particularly of the female sex. All these are factors of the social environment and movement of the population and are, consciously or unconsciously, under human control. In comparison with these, the influences of natural environment (climate, duration of sunshine, soil, etc.) are of minor importance.

The goal of a rational campaign against tuberculosis is still further to improve the social environment so that those infected by tuberculosis will not become the victims of the frank disease. Here one brings in treatment and therapeutics. If it were possible by means of genetic

study to determine what constitutional type affords the most protection against tuberculosis we should have another weapon in our armamentarium for attack upon the disease and be able to concentrate our measures further. It should never be the task of the physician, as defined by Hippocrates, to breed human types refractory to tuberculosis ignoring all other mental and physical hereditary qualities. If the 'norm of the ideal man' is not present to our senses, neither the statistician with his calculations of averages, nor the geneticist, cultivating a particular quality, nor the philosopher with his general ideas, will discover this norm. Such men as Rafael, Spinoza, Schiller, Mozart, and many others who died early from phthisis, are names we should hardly be prepared to forego in exchange for hereditary-biological qualifications and equality of breeding which might lead to the extinction of the type. So we are fortunate in knowing that it is possible, at least for most diseases, to proceed by way of environment and thus far to control disease. In this case we may be reasonably satisfied with what has already been achieved. By further extension of the same technique we may hope that tuberculosis will be brought almost to that vanishing-point which for leprosy, at least, in Europe, has already been attained.

Let us hope that this transformation of the environment which is our primary task, may not be disturbed again by war, hunger or pestilence. There is hardly any more sensitive measuring-rod than the reaction of mortality from tuberculosis in civilized states.

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ANTHROPOMETRIC STUDIES FROM McCORMICK FUND

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THE problems of statistical treatment of longitudinal data are complicated and much more serious than commonly recognized. When the investigation deals with a small, representative sample of a *public school* population—as to intelligence, nationality, and socio-economic status at the beginning of the study—the problem of procedures for analysis of the data is indeed serious. Criticisms by Meredith (4) published in a recent issue of HUMAN BIOLOGY of findings reported by us in *Healthy Growth* (3) appear to be due to some of these difficulties.

This paper will consider three issues involved in Meredith's criticism that "The trend of the evidence from other investigations definitely supports the opposite finding." (4, p. 570.) Two of these concern certain limiting conditions of the study and the third the treatment of the data.

Firstly, the study was set up with the specific purpose of improving the dietary and physical fitness of elementary school children in Joliet, Illinois, and of determining the influence of this improvement on their general development and adult status. A program of health education was carried on by pediatricians and health specialists with the children, their parents, and the school authorities over a four-year period. Evidence of improvement was definitely established. For example, at the conclusion of the program, a much larger proportion of the children were eating daily the types of food recommended by nutritionists for healthy growth (particularly milk, fruits, and vegetables) than before the study began, and more of them were rated by the pediatrician (after an examination without clothing) as in good or very good general physical condition with few remediable defects. These differences were statistically reliable. This factor of health improvement during the years covered by the measurements introduces a variable which should be considered in any critical evaluation of the anthropometric findings.

Secondly, the children in the group selected for the study were all from the same grade level—Grade III. They were not, of course, of the same age and intelligence. The oldest children were dull and backward and from the lowest socio-economic levels, while the youngest were unusually bright. In tables where either one or the other extreme in mental ability weights the growth data serious errors may be introduced, since numerous studies in this and other countries have shown some relation between mental and physical development during the growth period.[1] The mean physical measurements for 14 year olds quoted by Meredith from the Joliet Study are based on records of 16 dull and backward boys. The same subjects contributed largely to the measurements reported for 13 year olds.

Still another selective factor in the Joliet results is that of size. According to zones for stature classification designated by Baldwin, 20 per cent of the children included in the study were consistently classed as "short" and only 3 per cent as "tall" during the period of yearly measurements. At each age from 6 to 14 years the proportion of short boys was at least four times that of tall boys. Nationality was not, apparently, a factor in these results. It is possible that some mineral deficiency of the soil (particularly phosphorus) may have a bearing on the findings. However, in the opinion of agriculturalists[2] at the University of Illinois, the mineral deficiencies noted in their surveys of the locality are unlikely to have a retardative effect on animal growth.

These special conditions necessarily limit the validity of general comparisons of the Joliet findings with those obtained under different conditions. In our publications these points have been mentioned. The monograph quoted presents no direct comparisons with results from other investigations, with one exception—reports from Iowa Child Welfare Research Station at certain age levels. We stated that the trend of the Joliet growth findings for the years of middle and late childhood, on the whole, agree with those reported by others. However, at the 10 and 11 year levels our findings do not exhibit the deceleration noted by some investigators, and which was especially emphasized by Meredith (5, p. 95) as major minima in the course of growth of Iowa

[1] Among recent investigations Abernethy (1) reports a positive relation between mental test scores and physical measurements up to 14 years in a study from University of Chicago Laboratory School. Flory (2) concluded from his investigation of mentally deficient boys that boys who are mentally inferior are likewise physically inferior.

[2] Personal correspondence.

boys. Anthropometric measurements of the Joliet children were taken under the personal supervision of Baldwin as were many of those reported by Meredith. Both studies used repeated measurements. In an evaluation of the influence of health improvement on rate of growth, differences between these studies should be suggestive, when care is taken to consider variation in the types of subjects.

Further data are herein presented which bear out our former statement that the mean differences in growth with age of boys 8 to 12 or 13 years tends more to maxima than to minima between the 10th and 11th year for subjects included in the Joliet study. Measurements were made annually on 18 anthropometric traits for a period of four years but more extensive records are available on height and weight. The data on standing height are presented in Table 1 for 218 white boys, most of whom were measured for 5 or more consecutive years at an interval of 12 months. Age was verified from birth certificates.

TABLE 1

Mean trends in stature (cm.) of 218 white boys measured repeatedly during participation in a health education program

AGES	GROUP HAVING GENERAL PROGRAM				GROUP HAVING SPECIAL INSTRUCTION			
	N	Mean	S.D.	Diff.	*N*	Mean	S.D.	Diff.
8 (7-6 to 8-5)	90	123.9	5.65	..	24	125.5	4.55	..
9	135	128.9	5.54	5.0	93	129.8	5.79	4.3
10	168	134.0	5.93	5.1	130	134.5	6.22	4.7
11	168	139.3	6.13	5.3	125	139.2	6.49	4.7
12	90	143.6	6.33	4.3	58	143.4	6.02	4.2
13	14	147.3	6.54	3.7	14	147.3	8.24	3.9

The table gives the mean height in centimeters, the standard deviation, and the mean differences between successive ages. In the first section of the table every subject was measured at 10 and at 11 years of age and most of them at the preceding and following age levels. Section two of the table presents results from 138 boys who participated

in an intensive health education program, each boy being measured at least *three consecutive* times at yearly intervals.

It will be noted that differences between the mean height at 10 years (9-6 to 10-5) and at 11 years (10-6 to 11-5) do not indicate a decelerating growth rate at this period for the boys in the Joliet study. In both analyses the obtained gain between 10 and 11 years exceeds that of 8 to 9 years or 11 to 12 years. Figures beyond 12 years are weighted by backward subjects from the lower economic brackets. However, all differences are small and within the range of chance variations.

On the whole, the growth curves for boys in this study from the 8th through the 12th year approximate a straight line with little variation in any given year. The significance attached by us to the size of the gain between the 10th and 11th year lies in the possibility that an improved physical condition and general hygienic regimen during the previous years may have influenced the trend of these findings away from the diminution in rate reported by other investigators for this age interval.

While our results are somewhat at variance with those reported by many well-known investigators, they are in general agreement with the trend of findings from a number of studies from different sections of the country covering the economic period of our data. Table 2 briefly summarizes the differences in mean height at successive ages, for: 300 gifted California boys; 4630 public school boys of the Northeastern section of the United States; two Mid-western public school groups having repeated measurements taken in connection with a health program; and 1884 boys in the Laboratory School of The University of Chicago for whom information on the onset of puberty was available. These results give no evidence of outstanding minima in differential growth between the ages of 8 and 13 years, and the largest obtained difference in 3 of the 6 comparisons occurs between the 10th and 11th year.

In four of the studies cited measurements were made under the personal direction of Baldwin. With the exception of the Chicago data age was taken to the nearest birthday. The Chicago measurements were made on the birthday.

Children in most of these investigations were from a higher socio-economic level than those in the Joliet study. It has been frequently noted that children from well-to-do homes are better developed and

TABLE 2. *Average gain in stature (in.) of special groups of boys age 8-13 years*

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AGE	SINGLE MEASUREMENTS		REPEATED MEASUREMENTS				
	California Gifted[1]	Northeastern U.S.[2] "Old Americans"	Health Program Davenport, Iowa[3]	Health Program Oak Park, Illinois[4]	University of Chicago Laboratory School[5] Maturity Known	University of Chicago Laboratory School[5] Late Maturing	Joliet, Illinois[6]
	($N=300$)	($N=4630$)	($N=299$)	($N=522$)	($N=1884$)	($N=450$)	($N=215$)
8- 9	1.7	1.86	1.1	1.2	2.05	1.88	1.9
9-10	1.4	1.90	1.8	2.0	2.20	1.83	1.6
10-11	2.5	1.93	3.0	2.0	1.99	1.89	2.0
11-12	1.7	1.73	0.6	2.1	1.92	1.71	1.3
12-13	2.0	2.15	1.8	2.3	2.30	1.72	1.3

[1] Terman, Lewis M. *et al.* Genetic Studies of Genius. Stanford University, *California.* Stanford University Press, 1925, I, 145.

[2] Palmer, Carroll E., and Collins, Selwyn D. Variations in physique and growth of children in different geographic regions of the United States. U. S. Treasury Department, Public Health Service, Government Printing Office, *Washington, D. C.*, 1935. Reprint No. 1677. *Public Health Reports,* Vol. 50, No. 10, p. 12, Table 4.

[3] Baldwin, Bird T. The Physical Growth of Children from Birth to Maturity. *Iowa City,* State University of Iowa, 1921, 28-29. Also Terman, Lewis M. *et al., op. cit.,* 145.

[4] Elizabeth McCormick Memorial Fund. An Investigation of the Health of School Children: A two year study of the physical status of the children of the elementary schools of Oak Park, Illinois. (Under the direction of Maud A. Brown). *Chicago.* Elizabeth McCormick Memorial Fund, 1926, 88, Table 34.

[5] Richey, Herman G. The relation of accelerated, normal, and retarded puberty to the height and weight of school children. Monograph, Society for Research in Child Development, National Research Council, *Washington, D. C.,* 1937, Vol. II, No. 1, Serial no. 8, p. 42, Table XXXI and p. 14, Table VIII.

[6] Hardy, Martha Crumpton, and Hoefer, Carolyn H. Healthy Growth: A study of the influence of health education on growth and development of school children. *Chicago*: University of Chicago Press, 1936, 79.

tend to mature earlier than children from less favorable environments.[3] Hence comparisons of the Joliet growth findings with those of the late maturing boys of Chicago are particularly interesting. Inspection of the last two columns in Table 2 reveals a close correspondence with respect to mean differences for successive ages in the two studies. The findings in both instances show the gain from 10 to 11 years equal to that from 8 to 9 years, the trend favoring the 10 to 11 year level, and exceeding the 12 to 13 year interval.

A further point should be mentioned in connection with the "opposing findings" to the Joliet study. Palmer and Reed in an analysis of seriatim observations of a large number of individuals state that:

> "During the interval between the 10th and 11th year, particularly for boys that are above 52 or 53 inches in height, it is evident that the taller boys are growing more rapidly than the shorter ones. When the 11th year is reached, and thereafter as far as the data in this study extend, there is a definite and direct association between *annual increments of height* and *height itself.*" (6, pp. 325-327.) They conclude, "The adolescent acceleration in growth begins quite abruptly regardless of age, when boys reach a height of 52-53 inches and continues until a height of at least 60-61 inches is reached." (6, p. 333.)

According to the Baldwin-Wood standards few 10, 11, 12, and 13 year old boys in the Joliet study were "tall"—7 per cent, 7 per cent, 4 per cent, and 8 per cent, respectively—while a large proportion were "short"—28 per cent, 31 per cent, 29 per cent, and 48 per cent, for the respective ages. In the light of Palmer's findings the adolescent acceleration in growth should be less apparent in the Joliet boys (10-14 years) than in investigations in which tallness and shortness were more evenly distributed.

A distinctive advantage of longitudinal studies over cross-sectional is found in the greater reliability of patterns of growth from age to age. However, in common with many other investigators we have employed cross-sectional methods in our treatment of the data. Recently Shuttleworth (8, chapter 3, p. 216) has called attention to the fact that application of cross-sectional methods of analysis to seriatim measurements not only involves errors but means the loss of a most valuable part of the data. Further examination of our data corroborates his conclusions.

When the actual increments for successive ages of the Joliet boys were examined, the "relatively large" gain between 10 and 11 years

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[3] See reference (7), pp. 219-259.

previously obtained from differences between mean size at different ages is not apparent. By this procedure our data indicate a declining rate of growth after 8 years but the variations are small and stretch over the entire period included in the Joliet study. The lowest average increment is observed at the 11th to 12th year or the year following. This trend holds whether the results are from subjects followed without gaps from age 8 through 12 and later, or from all subjects measured at three or more successive ages; or again, whether the measurements were of stature or of other anthropometric traits.

Annual increments of growth in stature of boys in the Harvard longitudinal study reported by Meredith, show a general agreement with the cross-sectional studies cited by him in his criticism of the Joliet findings (4). His results on boys of North European and Italian nationality show the *lowest* mean increment of gain from the 8th to the 14th year to occur between the 10th and 11th birthday. The trend of his findings indicates a decelerating rate to the 11th year and an accelerating rate thereafter with relatively large increments at 8 to 9, and 13 to 14 year levels.

Table 3 presents the rate of gain in standing height from 8 to 13 years for Joliet subjects, based on an interval of twelve months between measurements of boys measured for three or more successive years (average 5 years). It was, of course, impossible to have every subject re-measured within an exact calendar year. For the great majority, the variation was not more than a few days but for others it was a month or more. Gains made in an eleven or thirteen month period have been corrected to twelve months. The table shows the median gains, the mean, and standard deviation for Joliet boys, the mean gains reported by Palmer and Reed for Hagerstown (Maryland) boys, and for boys of Beverley and Revere (Massachusetts), reported by Meredith.

Results from these three longitudinal studies on public school children are in agreement in showing a decrease in mean annual rates of growth in stature from age 8 years through the 10th year. The Hagerstown and Joliet data indicate a slight deceleration to the 12th year.

Detailed examination of the Joliet records gives no evidence of a definite maximum or minimum in the course of growth from 8 to 13 years, under the conditions of this investigation. Among boys measured consecutively from 8 or 9 years to 12 or 13 years the maximum gains were well distributed from age 8 to 12 years in stature and other measurements. Maxima were most frequent between the 9th to 10th

tend to mature earlier than children from less favorable environments.[3] Hence comparisons of the Joliet growth findings with those of the late maturing boys of Chicago are particularly interesting. Inspection of the last two columns in Table 2 reveals a close correspondence with respect to mean differences for successive ages in the two studies. The findings in both instances show the gain from 10 to 11 years equal to that from 8 to 9 years, the trend favoring the 10 to 11 year level, and exceeding the 12 to 13 year interval.

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or 10th to 11th year but at least 21 per cent of the boys made their largest gain in stature at each age interval included in the study. In breadth of shoulders no boy had his largest gain between 12 and 13 years but a large porportion had their minimum gain around 12 years (11-12 or 12-13). In stature 20 per cent or more made their smallest gain at

TABLE 3

Average annual increments of stature of boys measured repeatedly from 8-13 years

AGE	JOLIET, ILLINOIS				HAGERSTOWN, MARYLAND Palmer[1]		MASSACHUSETTS, NORTH EUROPEAN Meredith[2]	
	N	Median (*cm.*)	Mean (*cm.*)	S.D.	*N*	Mean (*in.*)	*N*	Mean (*cm.*)
8- 9	108	5.7	5.6	1.24	700	2.16	153	5.38
9-10	158	5.5	5.5	.90	813	2.00	155	5.13
10-11	169	5.3	5.3	.80	765	1.99	157	4.87
11-12	104	5.0	5.2	1.20	631	1.95	157	4.90
12-13	40	5.0	5.4	.72	409	2.23	157	5.74

[1] Palmer, Carroll E., and Reed, Lowell J. Anthropometric studies of individual growth: I. Age, height, and rate of growth in height, elementary school children. HUMAN BIOLOGY, 1935, Vol. 7, p. 323, Table I.

[2] Meredith, Howard V. Concerning a recently reported finding on the physical growth of boys. HUMAN BIOLOGY, 1937, Vol. 9, p. 569.

each age interval. The frequencies of maximum and minimum gain in height were approximately equal, at both the 8 to 9 and 11 to 12 age levels.

Analyses of the annual increments of growth of American-born white boys of mixed nationalities during participation in an extensive health education program have shown the largest mean increment in stature at 8 to 9 years with slightly smaller increments thereafter, as far as the data extend, but careful inspection of the individual records indicates that growth was spread fairly evenly over the years of this investigation. Some boys made their largest gains at one age and some

at another, no one age interval standing out as a particularly high or low point in the growth curve (8-13 years).

This statement is further borne out by the proportion of adult stature attained at different age levels. One hundred and fifteen of the boys measured in childhood were re-measured in early maturity (19-22 years, mean age 20 years). The results show that these boys had attained 65 per cent of their adult stature at 6 years, 71 per cent at 8 years, 77 per cent at 10 years, and 81 per cent at 12 years. The largest variation between successive intervals was less than 1 per cent, the percentage gain at 11 to 12 being smaller by this amount than that at the 10 to 11 year interval.

Growth trends from repeated measurements on stature of boys followed from age 8 years or earlier to 13 years are found to have the reliability of 1700 cases of cross-sectional data at each age. The 169 boys measured at 10 years and at later age levels give the equivalent of cross-sectional measurements on approximately 19,000 boys. Since each of these boys was measured at least three successive times at yearly intervals, we have the equivalent of more than 57,000 subjects.[4]

Detailed analyses of seriatim observations of 218 boys appear to warrant the conclusion that the pattern of growth from age to age (8-13 years) shows a fairly constant rate of growth during the years of middle and late childhood, with little indication of major maxima or minima peaks in the curves under the special conditions of the Joliet study.

SUMMARY

During participation in a health education program 218 white boys were measured repeatedly at yearly intervals. Marked improvement in physical condition and hygienic regimen was clearly evidenced during the period of the investigation. Analyses of the growth data by cross-sectional methods indicated relatively large gains at the 10 to 11 year interval, a finding which has been criticised by Meredith. When the emphasis is placed on growth increments rather than on differences in gross size at successive ages, the results suggest a slight deceleration from age 8 years as far as the data extend, but give no evidence of a significantly "high" or "low" point in the growth curve for ages 8 to 13 years under the special conditions of the health program. In comparing

[4] See reference (8), p. 23.

these results with those of other investigators, consideration must be given to the possible influence of health improvement on patterns of growth and other selective factors of this study.

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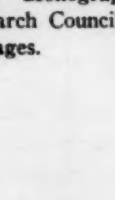

THE CORRELATION OF BONE VOLUME AND SOFT TISSUE VOLUME IN THE HUMAN FINGER TIP*

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AN ANALYSIS of data obtained in the course of observations on certain vascular phenomena of the human finger tip (Sodeman, '37; Turner, Burch and Sodeman, '37) has disclosed correlations between the volumes of bone and soft tissue of such high order as to be worthy of record. Following the terminology in the cited vascular studies, we arbitrarily define the finger tip, in terms of skin markings, as that portion of the digit distal to a plane passing through the major dorsal and palmar creases at the distal interphalangeal joint. All observations are limited to the right index finger. The method employed for the determination of the bone volume and total finger-tip volume is already described in detail (Sodeman). The total volume of the finger tip is determined by making a cast of the part in dental stone, the volume of the cast being then measured by mercury displacement. The bone volume is determined from antero-posterior and lateral areas and length of the phalangeal shadows in teleoroentgenograms, by the application of a formula experimentally calculated from comparisons of direct measurements of macerated phalanges with measurements of their radiographic shadows. The total soft-tissue volume is computed as the difference between total finger-tip volume and bone volume. The "bloodless" soft-tissue volume is obtained by subtracting the loss in finger-tip volume, determined plethysmographically, resulting from its drainage by elevation to 45 cm. above heart level followed by complete occlusion of the circulation (Turner, Burch and Sodeman).

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TABLE 1

Statistical constants for the finger-tip measurements in 26 normal adults

	MEAN	RANGE	STANDARD DEVIATION	COEFFICIENT OF VARIATION
Total volume ..	5269.25 ± 101.20 cu. mm.	4284 to 7511 cu. mm.	764.00 ± 71.43	14.499 ± 1.383%
Bone volume ...	734.61 ± 18.21 cu. mm.	538 to 1153 cu. mm.	137.40 ± 12.85	18.704 ± 1.698%
Total soft-tissue volume	4557.70 ± 83.34 cu. mm.	3582 to 6358 cu. mm.	629.00 ± 58.81	13.801 ± 1.315%
Length of bone segment	22.15 ± 0.19 mm.	19.2 to 27.2 mm.	1.40 ± 0.13	6.319 ± 0.593%

Observations were carried out on 26 normal adult males and 11 normal adolescent males. The statistical constants for the total volume, bone volume and total soft-tissue volume of the finger tip are recorded in Table 1. The correlation coefficient of bone volume and bloodless soft-tissue volume for the entire group is + 0.869 ± 0.027 (see Fig. 1), and for the adults alone + 0.850 ± 0.036. The correlation coefficients of bone volume and total finger-tip volume for the entire group and for the adults separately are + 0.899 ± 0.020 and + 0.835 ± 0.042, respectively.

COMMENT

The high correlation of bone volume and soft-tissue volume of the finger tip offers an interesting and exceptional contrast to the commonly very low correlation of anatomical structures. Most of the reported correlations involve anatomical structures which are not so closely related anatomically or physiologically. For example this is true of heart weight and brain weight, which give a correlation coefficient of only + 0.08 ± 0.08. However, in structures whose development and size are interdependent, such as the brain and its case, higher correlation is to be expected; and indeed this is true. Skull length and brain weight

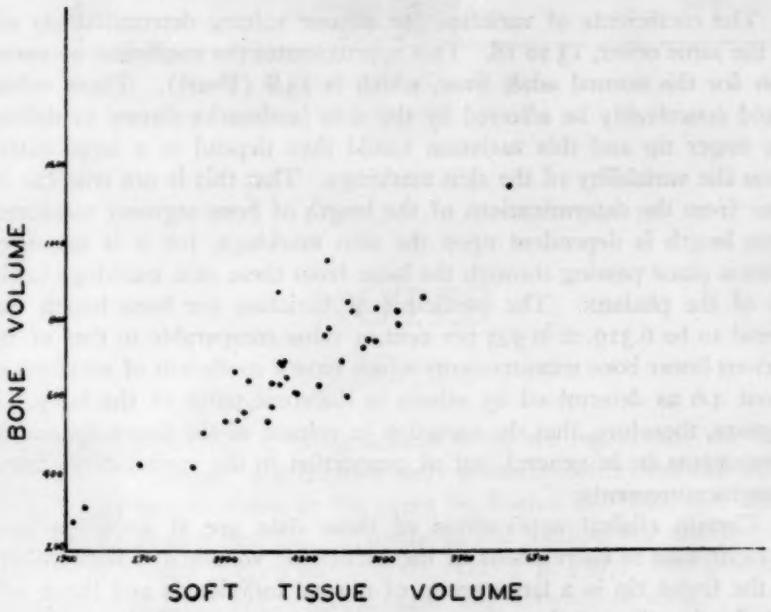

FIG. 1. RELATIONSHIP OF BONE VOLUME TO SOFT TISSUE VOLUME OF THE FINGER TIP IN 37 NORMAL SUBJECTS.

give a correlation coefficient of + 0.522 ± 0.028 (Pearl, '27). Because of the symmetrical development of the body, it is not surprising that bilaterally paired structures yield high correlations. This is exemplified in the correlation coefficient of the length of the first joint of the forefinger of the right and left hands, which was found to be + 0.925 ± 0.004 (Whiteley and Pearson, cited by Pearl).

In the instance of the structures we have correlated, namely bone volume and soft-tissue volume of the finger tip, the physiologic and anatomic relationships predetermined by development are not influenced appreciably by environmental factors, such as exercise and nutrition. Were one to attempt to correlate the volume of the humerus with the surrounding soft tissue, variable muscular development and deposition of fat would vary the volume of soft tissue independently of bone. These factors are minimized in the finger tip. The finger tip, therefore, would appear to yield a high correlation between bone and soft-tissue volumes simply because these two components are but little susceptible to independent volumetric fluctuations under changing nutritional or other states in the normal subject.

The coefficients of variation for all our volume determinations are of the same order, 13 to 18. This approximates the coefficient of variation for the normal adult liver, which is 14.8 (Pearl). These values could conceivably be affected by the skin landmarks chosen to delimit the finger tip and this variation would then depend to a large extent upon the variability of the skin markings. That this is not true can be seen from the determinations of the length of bone segment measured. This length is dependent upon the skin markings, for it is measured from a plane passing through the bone from these skin markings to the tip of the phalanx. The coefficient of variation for bone length was found to be 6.319 ± 0.593 per cent, a value comparable to that of the various linear bone measurements which have a coefficient of variation of about 4.0 as determined by others in different parts of the body. It appears, therefore, that the variation in volume of the finger tip and its components is, in general, out of proportion to the variations in linear bone measurements.

Certain clinical applications of these data are at once apparent. Comparisons of correlations of the soft-tissue volume and bone volume in the finger tip in a large group of normal individuals and those with arteriosclerosis or other diseases producing atrophy of the soft parts may be employed quantitatively to determine the degree of atrophy produced by such diseases.

We wish to thank Dr. Harold Cummins for his help in the preparation of this paper.

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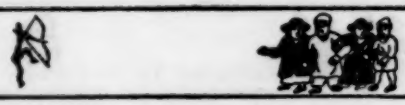
NOTES

ERRATUM

THROUGH a typographical error we regret that the word "clinical" was inadvertently substituted for "clerical" in the first footnote to the paper by Rachel M. Jenss and Nancy Bayley entitled "A mathematical method for studying the growth of a child" (HUMAN BIOLOGY, Vol. 9, p. 536). The assistance acknowledged from WPA project 4428 was *clerical,* not clinical.



RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Department of Biology, School of Hygiene and Public Health, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

I. EVOLUTION AND GENETICS

1. *Evolution—General Principles*

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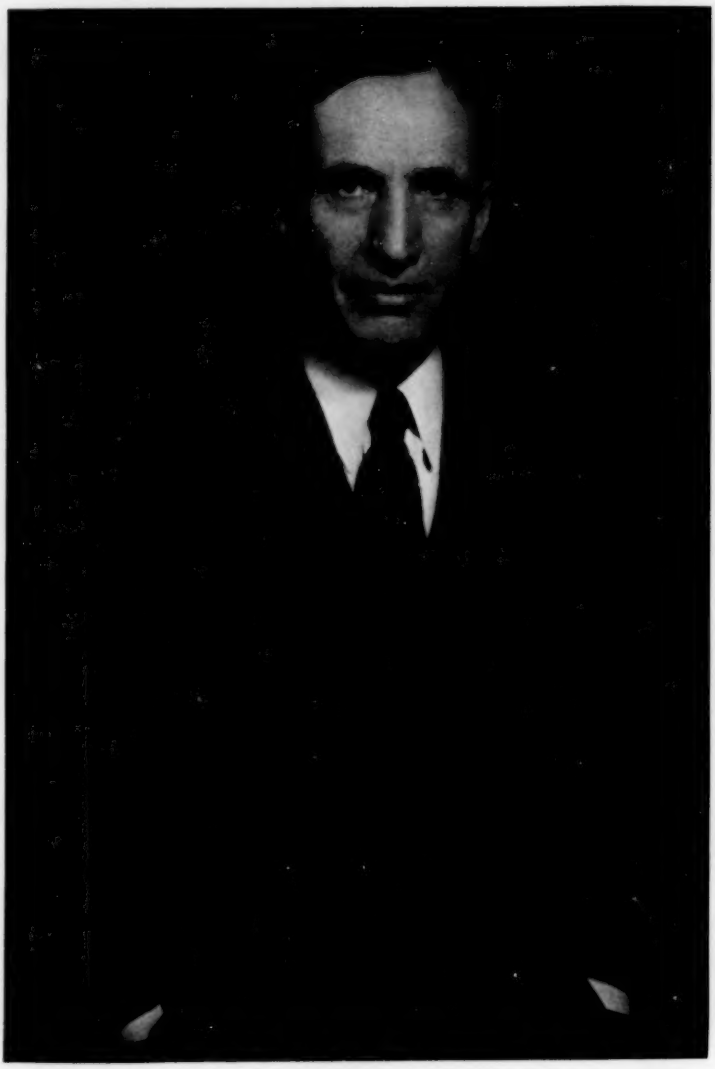

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